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RUPTURED INTESTINES

THE RESULT OF NON-PENETRATING TRAUMA

A CASE REPORT

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Severe and very often fatal injury to one or more of the abdominal viscera may result from direct blows or indirectly from explosions in the air or water. A sudden blow on the abdomen from a fist, a block of wood, or abdominal trauma from automobile accidents have resulted in serious and often fatal internal injuries. The numerous reports of people taken out of the wrecks of buildings during the London blitz, in apparently good condition, only to die within a very short time, the fatal results observed in many who received the concussions from depth bombs in the water, have but recently drawn our attention to this form of accident.

As revealed by operations or postmortems, the character and extent of these injuries have been amazing. Livers and spleens have been torn to shreds, the kidneys have been injured beyond repair, the bladder has been ruptured, intestines have been completely severed, the omentum has been torn asunder and blood vessels of vaious sizes severed. These, of course, represent the extremes of the injuries which are well nigh always fatal. There are other less severe injuries which after careful and painstaking examination backed by experience and good sound judgment, may be relieved by operation. Hemorrhage must be differentiated from shock, pain must be properly evaluated, and once the decision has been reached, the surgeon must back his judgment by immediate operation, consistent with sound surgical principles.

Evidences of severe injury are by no means always apparent immediately following the accident. Trauma may result to one or more portions of the gut, producing necrosis and sloughing some days later. Slight injuries to the solid organs may later result in abscesses. These, of course, are dealt with as they become apparent. Holes in the gut must be repaired or treated conservatively as the conditions indicate, and abscesses must be drained after they are well walled off.

The electrolytic fluids, of course, should be used freely, consistent with our established knowledge of the body's needs. The sulfonamides, the great gift to medicine and surgery, should be used locally, parenterally or per os freely. The recent increase in the manufacture of penicillin has enabled us to give our patients the benefits of this great life saving addition to our medical armanentarium.

The probable point or points of injury are, of course, always purely speculative, but having determined the place on the abdominal surface where the blow has been struck, one should with a limited degree of certainty be prepared to deal with specific organs. If the blow is over the upper abdominal cavity, the liver, spleen, pancreas, stomach, duodenum or transverse colon, one or more may be injured. A blow over the bladded may rupture that organ, while the intestines may be involved by any blow anywhere on the abdominal surface.

In the first twelve hours following injury, unless there is present the usual signs

and symptoms of severe hemorrhage, the surgical decision is very difficult, if not impossible. One is justified in "watchful waiting" and at the same time using all standard laboratory aids in addition to clinical observations. A board-like abdomen, unrelieved by opiates; distention, increase in the leukocytes with the well known differential count usually observed in infections, and finally air under one or both leaves of the diaphragm when the picture is taken in the sitting position, are enough evidences to justify the opening of the abdominal cavity.

The absence of signs of surface trauma such as swelling, ecchymosis or even skin tenderness, does not rule out by any means the possibility of a ruptured viscus. The full force of the blow can be transmitted to the intestines or one or more of the abdominal organs, and the skin even during convalescence may never have any signs of trauma.

CASE REPORT

A cabinet maker, 49 years old, a German Jewish refugee, while cutting a block of wood with a rip saw, was struck in the left lower abdomen when the saw broke. He was removed to his home where morphine was administered because of the intense pain from which he was suffering. He was later taken to the Schumpert Sanitarium where the opiate was repeated every four hours. An ice bag was also placed over the seat of injury. Twelve hours after the injury there was still no relief from pain. The abdomen was tender and board-like on palpation when the patient was seen shortly after the accident; now it was distended. There was no evidence of injury on the skin surface. There were no abrasions, no hemorrhages, no marks of any kind discernible even eighteen hours after the injury, nor was there ever any evidence of trauma to the skin at any time following the injury. The white count fourteen hours after the injury was 19,500 with 93 per cent polys. An x-ray showed no evidence of fracture of the pelvic bone, but there were considerable quantities of air under both leaves of the diaphragm when the x-ray was taken with the patient in a sitting position. These findings confirmed our suspicions of ruptured intestines.

A midline infra-umbilical incision was made. The abdominal cavity contained cloudy purulent fluid with the usual early inflammatory changes found in acute peritonitis. A single opening into the bowel, the circumference of a lead pencil, and straight longitudinal tears on either side of the opening through the serous membrane only, each about 1½ cm. in length, were found in the upper portion of the ileum. No other injuries were found after a careful examination of the abdominal contents. The opening was closed by a catgut purse-string suture and reinforced with a second line of interrupted sutures. The serous membrane tears were sutured with interrupted catgut sutures. The abdomen was closed without drainage after 10 grams of sulfanilamide were scattered throughout the abdominal cavity. The condition of the patient during the fifty-five minutes of the operation was excellent. The pulse varied between 94 and 100 per minute. At the beginning of the operation the blood pressure was 132/80. The patient left the operating table with a blood pressure of 150/90. Shortly after the patient was returned to his room 500 c.c. of citrated blood were administered. A continuous intravenous drip of 10 per cent saline and/or glucose was given, and every four hours 15 grains of sulfanilamide in 125 c.c. of distilled water were allowed to flow into the vein by using the same double bottle method used in administering citrated blood. Wangensteen suction was also instituted and a colon tube inserted from time to time in accordance with the judgment of the nurse on duty.

Although his general physical condition continued satisfactory, on the third day of the post-operative treatment he began to grow restless, incoherent in speech and finally become so violent that he had to be restrained. In spite of the watchful care of the nurse he got out of bed on the fourth day. Under forceful restraint four grains of sodium amytal were given by vein to quiet him. Six hours later 7½ grains were administered by vein, as opiates and hyoscine only made him more violent. At this time the urine showed 4+ albumin with a few hyaline, finely granular casts and red blood cells, with a trace of sugar. The blood showed a 10 mg. per 100 c.c. concentration of sulfanilamide. NPN was 56 mg. per 100 c.c. blood. As the cause of the delirium was apparent (the sulfanilamide concentration) the drug was discontinued. There was always an adequate amount of urine passed and the specific gravity was satisfactory throughout. His mania grew less, but was not entirely abated until the tenth day following the operation. At this time the sulfanilamide had entirely disappeared from the blood, the urine was negative and the bowels, which had moved from enema on the sixth day, were moving regularly. There was no abdominal distention at this time. A slight skin infection healed readily.

An interesting and unusual situation occurred during the height of the delirium. One night he called repeatedly, "Police, police." When I was contacted by the nurse, I instructed her to call the police. As soon as two police appeared at his bedside, he became quiet and dropped off to sleep. After he had recovered he remembered the incident clearly and explained that he thought he

was in a concentration camp in Germany and was being tortured by the Gestapo.

For some months after his release from the Sanitarium he complained of abdominal pain, which eventually disappeared. He is now back at work at his previous occupation.

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ASEPTIC ANTENATAL THROMBOPHLEBITIS (PHLEBOTHROMBOSIS)

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Antenatal thrombophlebitis is apparently a very rare condition, in contrast to puerperal thrombophlebitis (phlegmasia alba dolens), which is by no means infrequent. Westmann[1] sets the relative incidences at 0.1 and 5 per cent, but states no basis for the statistics.

There is almost nothing in the literature on the subject, and three of the 10 cases collected by Goldsborough,[2] when he reported an additional personal fatal case in 1904, are inadequately described. The disease is not mentioned in most textbooks of obstetrics. Stander,[3] in the 1941 edition of Williams' *Obstetrics*, refers to Goldsborough's report and adds, "Since then we have seen several additional cases," but supplies no details. Kahr,[4] in 1937, reported four cases of thrombosis of the deep veins of the lower extremities (as well as three instances of superficial thrombosis), and Maxwell[5] reported another case in 1943. I have no doubt that additional cases have occurred and have not been reported, and also that other cases have been reported but, as so often happens, cannot be identified by title when the literature is searched. At any rate, as matters now stand, the case I am reporting herewith seems to bring to 17 the number of formally reported cases, excluding the "several" cases mentioned but not described by Stander.

CASE REPORT

Mrs. W. R. P., a white primipara 36 years of age, was first seen during an uncomplicated, afebrile abortion at the end of the second month of a pregnancy which had been without special incident. Periods were missed for the next two months, but pain in the lower abdomen was present during the time menstruation would normally have occurred. Several examinations revealed a prolapsed and tender left ovary. Improvement occurred under a regimen of daily hot douches.

The patient menstruated for the first time since the abortion on August 20, 1942, but missed the September period. On the approximate date of the October period she bled slightly and passed a small clot. Although there was no recurrence of the bleeding she was kept in bed until November 24, 1942, that is, until after the date of the November period.

Her previous history contained no significant incident, and physical and routine laboratory examinations at this time revealed no abnormalities.

November 25, 1942, the day after the patient was first permitted to be ambulatory, she experienced pain in the calf and muscles of the left leg. She had no fever then or at any other time during the illnes. Examination revealed tenderness and redness along the entire course of the left saphenous vein, with pitting edema of the leg and thigh. A diagnosis of aseptic thrombophlebitis (phlebothrombosis) of the internal saphenous vein was made, and conservative therapy, including bed rest, elevation of the extremity, the application of heat and of an ace bandage, and a course of sulfathiazole, was instituted under the direction of Dr. Sidney Copland. When this regimen produced no improvement, sympathetic lumbar block with 1 per cent novocain was carried out on December 11, 1942. The pain in the limb was promptly relieved and the edema soon afterward disappeared. The patient was permitted out of bed with an elastic stocking on Decmeber 23. At this tme she weighed 134 pounds and the blood pressure was 110/70.

Pain in the calf and muscles of the right leg was experienced on February 14, 1943, and, as during the attack in the other leg, failed to respond to conservative therapy. Lumbar sympathetic block on this side was therefore carried out on February 26, again with prompt relief of pain and edema. Forty-eight hours after the operation she had a sudden sharp pain in the right thorax and presented the typical picture of an aseptic pulmonary infarct. She responded slowly to conservative therapy, including strapping of the chest, and was permitted to be ambulatory by March 13, 1943. From that time until the date of writing (October, 1944) she has had no recurrence of the vascular disturbance.

Pregnancy progressed smoothly until March 17, 1943; then bilateral edema of both lower extremities appeared and became slowly progressive until April 14, when edema of the face was also observed. The blood pressure, which had previously been stationary at 110/70, was now 116/76. The weight was 152 pounds. The urine contained no

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albumin or other abnormal constituents. A high-protein, salt-free diet was instituted.

During the next two weeks the edema of the legs did not progress but also it did not regress. The blood pressure remained at the same level. Pelvic examination on April 28 revealed the presentation to be vertex and the position ROA. The cervix was soft but not dilated. The child seemed small. Fetal heart tones of good quality were heard in the left lower quadrant. The fluid intake on this date was four and a half quarts and the output two and three-quarter quarts. The regimen already instituted was continued.

May 5, 1943, the patient weighed 154½ pounds and her blood pressure had risen to 130/90. The urine contained a trace of albumin. On the following day she complained of severe headache, her blood pressure rose to 170/100, and the urine contained 3 plus albumin.

She was at once hospitalized and was placed on a strict toxemia regimen. At the end of 48 hours her clinical condition was essentially the same as on admission, but the urinary albumin had not decreased and her blood pressure had risen to 200/100. Labor was therefore induced without delay by the serial administration of small doses of pitocin. Pains ensued promptly, and delivery of a five-pound male child was effected without difficulty 11 hours later by low forceps and episiotomy. Hemorrhage was minimal. When morphine gr. 1/4 was administered after the delivery of the child, the patient's respirations fell promptly to eight per minute, but rose to the normal level after the administration of oxygen.

The child was normal in all respects except for a slight hypospadias. Soon after birth, however, he became cyanotic, and attacks of cyanosis persisted for almost a week and were sometimes alarming. Feeding also presented difficulties, but eventually an appropriate formula was found and he was discharged from the hospital in good condition, after a satisfactory gain in weight.

The mother's convalescence was smooth except for the development of a moderate lymphangitis and phlebitis of the left upper extremity, clearly originating in chemical irritation following infusion. When she left the hospital on the fifteenth postpartal day her blood pressure was 120/80, she weighed 134½ pounds, and the urine was free of albumin. A moderate erosion of the cervix was treated by cauterization several times in the next three months.

The patient has been observed at intervals to date (October, 1944) and has presented no abnormality of any sort, including, as already stated, no recurrence of the vascular abnormalities.

To complete the history, it should be stated that laboratory examinations threw no light on this case. The blood serologic reactions were negative. Complete hematologic study three weeks before delivery revealed the following data: Coagulation time two minutes 45 seconds. Bleeding time four minutes 10 seconds. Clot reaction good. Red blood cells 4,580,000, white blood cells 9,850 per cu. mm. Hemoglobin 80 per cent (12.5 gm.). The differential values were within normal range except for a slight shift to the left of neutrophiles. The prothrombin concentration was 115 per cent.

COMMENT

The number of reported cases (17 including the one reported herewith) is too small for significant analysis, but certain data derived from them might be mentioned. Both unilateral and bilateral involvement occurred and both the saphenous and femoral veins, with their branches, were affected. Fever was seldom a part of the picture, and the diagnosis of aseptic thrombophlebitis (phlebothrombosis) was therefore usually warranted, as in my personal case. Recovery was usually complete, though all patients did not go to term, but Goldsborough's case ended fatally, and Bacon's[6] patient, one of the few with fever, continued to suffer from swelling and tenderness in the leg when she began to walk five weeks after delivery and about 60 days after the onset of phlebitis.

The case reported herewith differs from most reported cases in the early onset of the complication, at the end of the third month of pregnancy, when the uterus, although enlarged, is not yet large enough to cause much pressure on the pelvic structures. The patient presented no evidence of focal infection or of any other abnormality which might have been related to the vascular condition. The toxemia, which is listed as a possible factor in some of the reported cases, occurred about four months after the first attack of thrombophlebitis and more than a month after the recurrent attack in the other leg, which seems to rule it out as a cause. Indeed, the only factor in the whole history which can be related to the development of thrombophlebitis is the prolonged period of rest in bed, to avert a threatened abortion, which terminated exactly 24 hours before the vascular condition first became evident. Stasis due to prolonged bed rest is, of course, an important factor in the development of postoperative thrombosis and thrombophlebitis.

In most of the reported cases either there is no evident etiologic factor, or the cause advanced is applicable only to the special case or is entirely unreasonable. Browne (cited by Maxwell) mentioned varicose veins as a predisposing cause, and they frequently are responsible for superficial venous thromboses in pregnant women. But many pregnant women, as in this case, do not present varicosities, and the explanation is therefore not generally applicable. In Maxwell's own case the only variation from the usual was the fact that the pregnancy was a twin-gestation, but the author does not seem to regard this as an explanation. The patient had a bilateral involvement of the great saphenous vein and its lateral branches 39 days before delivery, which occurred 14 days before the expected date; recovery was complete before she left the hospital.

Bacon's patient, already mentioned, developed uterine contractions soon after a fall on the ice in the thirty-third week of pregnancy, and 10 days later developed fever and great pain and edema of the left leg. The author's suggestion that "the long-continued uterine contractions may have dislodged placental masses that formed rudimentary emboli, or perhaps altered the blood composition," is a concept as speculative as it is generally inapplicable, but it was advanced in 1903. Westmann states that it is "not a mere hypothesis" to assume that women who incline to varicose veins, thrombosis and embolism must be considered as possessing inferior cardiovascular systems, most probably the result of endocrine insufficiency, but advances no proof for the theory and reports no cases of antenatal thrombophlebitis.

Veal and Hussey,[7] whose discussion is also general, grant the responsibility of such factors as the size and position of the uterus, the length and mobility of the supporting ligaments, the size of the pelvis, and the tone and development of the abdominal musculature in the production of abnormalities of the venous circulation in the lower extremities during pregnancy. After a study of popliteal venous pressures in pregnant women during exercise they concluded that postural dependent edema and varicose veins of the lower extremities are due to localized obstruction of the deep veins. This theory explains individual cases of this sort, and furnishes the only reasonable explanation for unilateral edema and varicosities. It is quite possible that localized obstruction of the deep veins may prove the explanation of antenatal thrombophlebitis, though I am not aware that any case has yet been studied from this point of view.

In Goldsborough's case the etiologic factor seems clearcut. The patient, a 29 year old working woman, in an endeavor to conceal her pregnant state (it should be remembered that this case was reported in 1904), wore a very tight, heavily boned corset. She was first seen in the seventh month of gestation, a week after the development of edema and swelling of the left leg and four days after the development of almost continuous vomiting. The left leg presented an enormous symmetrical pitting edema extending from the toes to the groin, and there was considerable abdominal distention. The uterus was displaced to the left. Urinalysis was essentially negative.

The patient's vomiting could not be controlled by any method then available and bag induction of labor was therefore carried out. The bag burst 10 hours after its introduction, but the patient went into labor, which was eventually terminated by manual dilatation of the cervix, version and extraction performed forcibly and with much difficulty in a tetanically contracted uterus, and manual removal of the placenta. The child was born dead, and the patient, who was badly shocked and had lost a large amount of blood, died 15 hours after delivery.

Postmortem examination showed marked degeneration of the liver cells and of the kidneys, especially in the convoluted tubules. The patient had had a large amount of chloroform, which perhaps explains these findings, on the basis of the liver-kidney syndrome.

The lumen of the left common iliac vein was completely occluded at the point of its crossing by the right common iliac artery. The vessel from this point downward was filled with a thrombosed mass which extended into the external iliac vein and thence into the femoral and saphenous veins; above the point of compression it was perfectly normal. Histologic examination showed practically no change in the vessel walls except for the absence of the endothelial lining. Signs of marked inflammatory reaction were lacking. The entire lumen was merely filled by blood clot firmly adherent to the wall. Cultures from the heart blood and from the thrombus were sterile, and bacteria could not be demonstrated by any method.

The author speculated, with complete reasonableness, that the origin of the thrombosis in this case was pressure on the retroperitoneal structures by the patient's heavy corset. As the enlarging uterus was forced backward and downward against the bodies of the lumbar vertebrae and the structures in front of them, it interfered with the return of the blood from the lower extremities, particularly at the crossing of the right iliac artery by the left common iliac vein. As the pressure increased, the iliac vein eventually became compressed between the artery in front and the vertebral column behind, with the result that the lumen became completely obliterated, with terminal stagnation, coagulation and thrombosis. The explanation is adequate, but the case is unique.

The cause of thrombophlebitis in nonpregnant subjects is still a matter of dispute, and it is scarcely surprising that no universal cause has been advanced for the small group of cases reported in pregnant women. The relief obtained by lumbar sympathetic block in the case reported herewith supports the concept of a possible vasospastic background. It is also possible that this particular case (and perhaps other cases) is an instance of so-called primary idiopathic thrombophlebitis of the recurrent type, which was formerly called thrombophlebitis migrans. The subject was thoroughly reviewed by Barker[8] in 1936. In a report of 79 cases observed at the Mayo Clinic, 40 of which were of the recurrent type, acute infarction occurred in 12 instances, as it did in my personal case, and five patients in the group eventually died of pulmonary embolism.

Although the only fatality in the reported instances of antenatal thrombophlebitis seems to be Goldsborough's case, in which the circumstances are obviously unique, it is well to emphasize that this is always a potentially serious condition. Labor, as several authors have pointed out, may provide sufficient impetus to cause liberation of a thrombus in the affected vein or veins, though, happily this does not seem to have occurred in any of the reported cases. The possibility, however, should be borne in mind, and all manipulations in such patients should be extremely cautious, with the recollection that fatal pulmonary embolism may be the end-result of carelessness in this regard.

SUMMARY

A case of aseptic antenatal thrombophlebitis (phlebothrombosis) is reported, and is apparently the seventeenth to be recorded with any detail in the literature. No universally applicable explanation has yet been advanced for the condition. Although only one of the recorded cases seems to have terminated fatally, the complication is potentially serious and all manipulations in such patients should be very gentle.

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CERTAIN ETIOLOGIC FACTORS CONCERNED WITH HEADACHE*

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The number of etiologic factors productive of the prevalent symptom, headache, is infinite and the real cause is difficult to determine. Consequently, specific treatment remains a problem. Fortunately, in most instances, no treatment is necessary or palliative measures are sufficient to relieve the discomfort. However, the patient with the chief complaint of headache offers a distinct challenge to the diagnostician's acumen. Of some importance in the determination of the cause of cephalalgia is an understanding of the nerve supply to the head and the known mechanisms by which head pain is produced. Therefore, these and other points will be briefly discussed as a possible method of approach in the determination of the cause of headache.

NERVES INVOLVED IN HEAD PAINS AND THEIR DISTRIBUTION

Extracranially, from all areas of the head anterior to a line drawn vertically in front of the ears, pain is mediated by way of the three branches of the fifth cranial nerve. Behind this line sensation is transmitted by the upper three cervical nerves. In addition, the ninth and tenth cranial nerves are supposed to have some sensory terminations in the external auditory canal.

In relation to head pain and headaches in general the eyes should be considered as a possible factor. The structures of the eyes are innervated by the ophthalmic division of the trigeminal nerve. Sensation originating in the cornea is localized accurately. However, it is more difficult to determine the source of pain when it is within the deeper structures. It has been shown[1] that traction on the ocular muscles and ciliary body produces pain deep within the orbit which may radiate over the entire distribution of the first branch of the trigeminal nerve. Induced imbalance of the extraocular muscles, if prolonged, will cause nervous tension followed by headache. This headache is associated with abnormal myograms of the scalp and posterior cervical muscles. As a matter of fact, any inflammatory condition of the eyes, glaucoma, astigmatism, hypermetropia, or abnormal accommodation, may in a similar fashion produce tension in the muscles of the head and neck with subsequent headache. On the other hand, myopia does not often produce pain or headache of this type.

Another extracranial source of headache is the nasal cavity. It has been found that the sinus mucosa has a low order of pain sensitivity.[2] Stimulation of these mucous membranes by various means never causes pain in the neck or back of the head. In general, severe pain occurs only with inflammation and engorgement of the turbinates, sinus ostia, nasofrontal duct, and superior nasal spaces. It should be noted further that pain originating from the nasal structures involves primarily the distribution of the maxillary or ophthalmic branch of the fifth cranial nerve, which, under certain conditions, may spread to other parts of the head.

Within the cranial vault, pain originating above the cerebral tentorium is mediated over the ophthalmic branch of the trigeminal nerve; the other two divisions of the fifth cranial nerve have no known intracranial terminations. Below the tentorium, pain sensation is transmitted along branches of the ninth and tenth cranial nerves and of the second and third cervical nerves. From no part of the head is there evidence to indicate that afferent fibers of the sympathetic system transmit pain or other sensations arising in consciousness.

Furthermore, all structures external to the bone of the cranial vault, including the vascular system, are sensitive in varying degrees[3]. The bone, the pia-arachnoid, the brain substance itself, the ventricular walls and the choroid plexus are, however, insensitive to painful stimuli. The dura mater is only painful when stimulated adjacent to the main arteries or upon extreme pressure. A few of the venous sinuses and connecting extremities of cerebral veins are likewise

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pain sensitive. The arteries in forming the circle of Willis and for about one-third of their length distally give rise to pain. The middle meningeal artery is extremely sensitive to pain throughout most of its course.

In brief, it is known that extracranial pain is usually well localized and similar in character to cutaneous pain elsewhere in the body. However, pain originating extracranially from the eyes and sinuses assumes the characteristics of visceral pain elsewhere and is of a disagreeable nature, radiates widely and is difficult to localize with accuracy. The same remarks apply to pain originating within the cranial vault. Thus, it is well to recall that intracranial disease in the back of the head may refer pain to the frontal areas due to the prolongation backwards of a branch of the ophthalmic division of the fifth nerve.

MECHANISMS BY WHICH HEAD PAIN IS PRODUCED

Several mechanisms have been demonstrated by which intracranial pain is produced[1]. First of all, there may be traction or stretch placed upon the dura mater or vascular system as a cause of pain. Thus, after removal of cerebrospinal fluid, stretch is undoubtedly placed on the cerebral veins as they enter the sagittal and other venous sinuses. Proof of this mechanism is shown by the relief of such pain when the fluid volume has been restored or the head has been tilted downward to one side. By both of these means traction is reduced. Similarly, in expanding lesions such as tumors, hematomas, aneurysms, hemorrhage or edema, stretch may be applied to the arteries at the base of the brain or to the middle meningeal artery with consequent stimulation of pain sensitive structures.

A second obvious mechanism for head pain is direct pressure of a lesion upon a nerve. However, this is probably of little importance until late in the course of a disease because of the capacity of the brain to shift its position slowly in a fluid matrix. A third incitement for pain is direct stimulation of pain sensitive endings by bacterial agents or their products and possibly other toxins.

A fourth mechanism for inducing headache intracranially is concerned with abnormal dilatation or loss of tone of the several pain sensitive arteries. Several factors need to be considered for a partial understanding of this phenomenon. It should be noted that arterial dilatation elsewhere in the body is seldom painful. This difference may possibly be explained on the basis of the more rigid confines of the scalp and cranial cavity. More germane to an understanding of this mechanism is the relationship of the blood pressure and the cerebrospinal fluid pressure to each other and to the tone of the arterial walls. These relationships have recently been vividly demonstrated by several investigators[4, 5] who employed strong vasodilating drugs, such as histamine or nitrates, experimentally.

The intravenous administration of histamine causes rapid dilatation of the arterioles of the body, including those of the cranium. Headache does not develop concomitantly with this widespread loss of tone. At first there is an abrupt decrease of the systemic blood pressure and an associated rise of the cerebrospinal fluid pressure. The latter is apparently sufficient to maintain some arterial tone. Shortly afterward, however, the blood pressure rises rapidly to its original level, as the systemic arteries regain their tone, which they do long before those of the cerebral circulation. As the systemic blood pressure returns to its previous level, the cerebrospinal fluid pressure rapidly declines; this removes this support to the already distended cerebral arteries. Thereafter, with each wave of the pulse, the typical throbbing headache is experienced and gradually declines in intensity over a period of six to eight minutes as the cerebral arteries gradually regain their normal tone. A similar sequence of events is associated with the headache induced by nitroglycerin.

While on the subject of headaches originating from vascular structures, it might be well here to discuss briefly a few points about migraine and hypertensive headaches not associated with heart failure or encephalopathy. These headaches are like-

wise due to arterial dilatation[4], but the arteries most prominently involved are extracranial, that is, the branches of the external carotid artery. Furthermore, migraine and hypertensive headaches are not affected by changes of the blood pressure or the cerebrospinal fluid pressure. As a matter of fact, it has been repeatedly demonstrated that the administration of ergotamine tartrate will abort or relieve the headaches of migraine and hypertension. In so doing, this drug acts specifically upon the branches of the external carotid arteries and at the same time induces a further rise in blood pressure.

Although several factors and mechanisms concerned with headaches have been discussed, it would be misleading to give the impression that the site or nature of organic disease can be readily diagnosed in the light of these concepts. Headaches of organic origin are more usually a combination of two or more of these mechanisms.

In the case of expanding lesions[6], such as brain tumors, aneurysms or hematomas, the character of the pain varies widely even in the same person. Sometimes, it may be throbbing and therefore seemingly due to vascular distention as a localized process. Again, it may be intensified by certain movements of the head; this suggests a traction origin. On other occasions the pain may be sharp and lancinating which may be due to direct pressure on a nerve trunk.

It is the general impression that the increased cerebrospinal fluid pressure usually associated with expanding lesions is the cause of the concomitant headache. Statistics refuting this statement have been reported.[6] Of 65 cases of brain tumor with headache, 49 had increased cerebrospinal fluid pressure. In another series[7] of 121 cases, 63 per cent of the patients had pressure over 200 mm. of water and many of these had no headache. Furthermore, as noted previously,[4, 8] many headaches of vascular origin may be relieved by increasing the cerebrospinal fluid pressure. However, if there is a block to the flow of spinal fluid, the incidence of headaches becomes practically 100 per cent. The mechanism of headaches in such cases is clearly that of traction.

The localization of pain associated with brain tumors or other expanding lesions is not too helpful in diagnosing the site of the neoplasm.[6] Frontal pain is experienced in 95 per cent of patients with supratentorial tumors; 75 per cent of patients with infratentorial tumors likewise have frontal pain. Pain and spasm in the back of the neck were found in 22 per cent of patients with supratentorial tumors and 45 per cent with infratentorial growths. Of 66 patients with unilateral headaches, the tumor was found on the opposite side in 15 instances. It seems obvious, therefore, that in the diagnosis of a growing lesion within the cranium the facts concerning the pain are of little help in general.

Another type of headache is that occurring subsequent to brain concussion. The differentiation between those of purely organic and those of psychogenic origin seems to be a matter of personal opinion in most instances. Denny-Brown[9], in experiments on animals, showed that during the period of unconsciousness, characteristic generalized patterns appeared in the electro-encephalogram, even though microscopic evidence of damaged tissue was seldom found at autopsy. Persistent deviations from the normal patterns may eventually be interpreted as a specific sign of post-concussional pathologic condition.

One of the characteristics of the post-concussional syndrome is the paucity of findings pointing to a known organic lesion. The incidence of disability varies from 20 to 80 per cent. In the presence of severe brain damage, it is noteworthy that comparable symptoms develop in only 10 per cent of cases. It is apparent that underlying the development of the post-concussional syndrome, there is a preceding history of anxiety and emotional instability associated with poor physical development and vasomotor over-reaction. All symptoms and signs of the various headache mechanisms are found in this syndrome. The pain is often made worse by excitement,

fatigue, exertion, changes in the weather, alcohol and various movements of the head. It is frequently accompanied by dizziness, nausea and vomiting. Diagnosis, therefore, still rests on the exclusion of other factors in conjunction with a thorough history, physical examination and determination of the psychic make-up.

Before concluding, another factor which seems important in the approach to the determination of the cause of headaches should be mentioned. This is the time of onset of the headache within the 24 hour period. Headache, associated with the following conditions, is prone to awaken the patient or is present when he arises in the morning: (1) expanding intracranial lesion; (2) maxillary or frontal sinusitis; (3) hypertension with early renal failure or encephalopathy and perhaps other conditions in which edema or dehydration of the brain occurs, such as in a "hangover," in hyperinsulinism or after the excessive administration of hypnotics; (4) cervical arthritis associated with strained positions in sleep and (5) histaminic cephalalgia.

Headaches occurring late in the afternoon or evening may be caused by various factors. The involvement of the ethmoid and the sphenoid sinuses is thought to produce headache late in the day. Fatigue from any cause, more specifically that induced by diseases of the eyes, toxins, such as tobacco and, most important, nervous tension, may produce headaches which seem to occur more frequently as the day progresses. Headaches of these origins, excluding migraine, are more characteristically located either in the vertex of the skull, which is a site of pain seldom referred to in organic disease, or in the back of the head and neck. The latter headache or pain is caused by persistent tension of the muscles of the neck and scalp associated with abnormal myograms.[10] Palpation of this area reveals definite spasticity of the cervical muscles, and here it must be recalled that with intracranial expanding lesions, as well as with arthritis of the cervical spine, there may be spasticity of the same group of muscles.

CONCLUSION

In the determination of the cause of headache the essential fact to be remembered is that headache is merely a symptom which, in most instances, gives little information as to its exciting cause. The benign or malignant stimulant of the pain sensitive structures can rarely be differentiated by the character or distribution of the pain itself. Nor does a knowledge of the pain mechanism concerned always provide a specific etiologic diagnosis. To serve the patient faithfully, therefore, a thorough evaluation of his physical and mental condition is essential.

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TREATMENT OF AMEBIASIS

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DEFINITION

Amebiasis is defined as the state of being infected with the protozoan organism known as *Endamoeba histolytica* and the term includes all of the clinical pictures caused by the invasion of this organism. Amebiasis embraces the signs and symptoms in mild infections in which vague

gastrointestinal symptomatology is present; those present accompanied by severe enteritis; those present in acute amebic dysentery; and those present in complications from invasion of the liver, lungs, brain, skin or other organs by *Endamoeba histolytica*, as well as carrier states.

HISTORY

The first accurate description of the causative organism we owe to Lösch who in 1875 in St. Petersburg, Russia, studied an undoubted case of amebic dysentery with relapses and found the organism in the stool. He named the organism *Amoeba coli*. Shaudinn in 1903 accepted the name for the genus previously proposed by Casagrandi, but named his pathogenic species *Endamoeba histolytica* and the non-pathogenic *Endamoeba coli*. Sir William Osler was the first person in the United States to demonstrate the amebae in a case of dysentery and liver abscess.

DISTRIBUTION

Amebiasis is world-wide in distribution. It can no longer be considered a tropical disease. In some districts in the temperate zone where sanitary conditions are poor, the incidence may be almost as high as in tropical countries. In the United States it has been conservatively estimated that from 5 to 10 per cent of the population harbor the organism. Since only a relatively small percentage of infected patients suffer from the disease, the number of asymptomatic carriers is exceedinly large.

COMPLICATIONS AND SEQUELS

Amebic abscess of the liver is the most important and most frequent complication of amebiasis, and usually occurs after attacks of diarrhea or dysentery, but may occur in a carrier who has never had such attacks. Amebic abscess, abscess of the lung, brain, spleen and other organs have been repeatedly reported. Perforation of amebic ulcer of the intestine sometimes occurs with a resulting peritonitis, and the erosion of a blood vessel by such ulcerations with the consequent production of fatal hemorrhage from the bowel has been reported by several investigators.

The most common sequels of acute or chronic amebic dysentery are contractures of the large intestine by adhesions and cicatrices which sometimes cause obstruction of the bowel, or more frequently marked constipation due to the decrease in the caliber of the bowel.

PHARMACOLOGY OF AMEBICIDES

At this time there is a need for a careful appraisal of all available amebicides because of their extensive use by the armed forces. Amebicidal drugs may be classified as follows:

I. The Emetine Group
   (a) Ipecac
   (b) Emetine hydrochloride
   (c) Emetine bismuth iodide
II. The Oxyquinoline Derivatives
   (a) Chiniofon
   (b) Vioform
   (c) Iodoquin
   (d) Diodoquin
III. The Organic Pentavalent Arsenicals
   (a) Carbarsone
   (b) Treparsol
   (c) Acetarsone
IV. Miscellaneous Drugs
   (a) Succinylsulfathiazole
   (b) Bismuth compounds

The physician has at his disposal six potent amebicidal agents: emetine, carbarsone, chiniofon, vioform, iodoquin and diodoquin. These six drugs will be discussed and the less important drugs will be omitted in this paper. Consideration of your time and patience forbids detailed enumeration of all drugs employed in the treatment of amebiasis.

EMETINE

Emetine was first described by Pelletier, the discoverer of quinine in cinchona bark. Bardsley in 1829, was apparently the first to use emetine in diarrheas and dysenteries, and Rogers in 1912 obtained good effects from the drug in amebic dysentery and hepatitis.

Emetine is an alkaloid of ipecacaunha. The hydrochloride salt is used because of its greater solubility. For many years eme-

tine hydrochloride was regarded as a specific drug for amebiasis, but it is now known that while it is most efficient in controlling the symptoms of amebic dysentery, it is not capable of eliminating amebic infection when employed alone except in a small proportion of cases. Craig[1] has pointed out that the disappearance of symptoms does not indicate elimination of the infection in patients treated with emetine. For this reason emetine hydrochloride should not be employed in the treatment of amebiasis to eliminate the infection but only to control the symptoms of severe diarrhea, after which one or the other of the several amebicides can be used.

Emetine is a general protoplasmic poison and exerts a cumulative toxic action since it is excreted or detoxified slowly. The myotoxic action is particularly evident on heart muscle in which it can cause a severe acute degenerative myocarditis which may result in sudden cardiac failure or death. Emetine is the most toxic of drugs used in amebiasis. There is no known antidote for emetine.

Nausea and vomiting may occur after emetine. Severe diarrhea is not uncommon and the stools may contain blood.

Emetine has a direct lethal action on *Endamoeba histolytica*. The drug is more effective against the motile forms than against the cysts. Concentrations of emetine necessary to kill cystic forms cannot be safely obtained in man.

CHINIOFON

Chiniofon is an iodoxyquinoline derivative. It was introduced in 1921 under the name of yatren. Anayodin is a propietary preparation of chiniofon.

Chiniofon depends on iodine for its amebicidal properties. It contains 26 to 28 per cent iodine. The action of this drug is apparently a direct one upon the motile forms as well as cyst forms. Its proper administration is followed by elimination of the infection in most cases. In therapeutic dosage it occasionally gives rise to severe diarrhea. Chiniofon is non-toxic in therapeutic doses. Given intravenously the drug has caused death, but there is no need or excuse for using it by this route. This is one of the safest drugs than can be used.

VIOFORM

Vioform is iodochlorhydroxyquinoline. Vioform, like chiniofon, is directly amebicidal due to its iodine content. Vioform contains 37.5 to 41.5 per cent iodine and 11 per cent chlorine. The toxicity is greater than that of chiniofon, but its toxicity is almost negligible. In large doses it produces fatty infiltration of the liver in rabbits.

CARBARSONE

Carbarsone is an organic pentavalent arsenical. Carbarsone is directly amebicidal by virtue of its arsenic content. This drug contains 28.5 per cent arsenic and acts directly on the motile forms as well as cysts. The toxic symptoms are those of arsenical poisoning. Reed[2] is of the opinion that carbarsone is the best of all amebicidal drugs. Craig[6] (1934) places it second to chiniofon. It is the most potent and least toxic arsenical amebicide, is as effective as any other single agent, and rarely causes serious toxic effects.

DIODOQUIN

Diodoquin is a new oxyquinoline compound. It contains approximately 64 per cent iodine. The action of diodoquin upon *Endamoeba histolytica* presumably is directly dependent upon the iodine content of the drug. The dosage varies with the severity of the infection. No toxic symptoms have been reported save a heaache in rare instances.

Forty-one cases were treated by Hummel[3] with diodoquin. This drug was found to relieve the colonic and nervous symptoms in a comparatively short time. Relapses were not observed and toxic effects were not encountered. Trophozoites and cysts disappeared in all cases from 10 to 15 days from the time treatment was begun. The therapeutic effects on the colon and rectum were observed by frequent sigmoidoscopic and stool examination during and after treatment. From his work he concludes that this new oxyquinoline compound is a valuable drug, fulfilling the criteria of the ideal amebicide more than any other preparation available at the present time.

The statement has been made that diodoquin is recovered quantitatively in the stool and even with enormous dosage no absorption takes place. David,[4] on the other hand, found diodoquin to be occasionally lethal in animals and capable of causing liver damage and hence one can assume that absorption is possible after oral administration under certain conditions. This was proved by studying the blood iodine levels in healthy human subjects given therapeutic doses of either vioform or diodoquin. From their results they found that a greater rise in blood iodine occurred with vioform than after diodoquin. More consistent absorption of vioform was noted. No symptoms of iodine poison were observed. Diodoquin is extremely insoluble in water, dilute acids or alkalies. It is believed that toxicity of an insoluble type of compound such as diodoquin may depend more on hyperacidity or intestinal stasis than on the actual amount of drug administered. When such conditions are encountered the potential danger of toxicity is greater with diodoquin, since it contains 63.9 per cent iodine as compared with 41.5 per cent iodine in vioform. Apparently the unreliability and irregularity of the absorption of diodoquin after oral administration is a major handicap for its uncontrollable use. On the basis of their results they believe that vioform will prove a more reliable amebicide and less likely, due to its less variable absorption, to cause toxic symptoms. The use of either of these compounds in the prophylaxis of amebiasis must be rigidly controlled and should not be carried out as extremely as is done in the prophylaxis of malaria. Any procedure intended to safeguard the health of troops and workers in amoeba infested areas is worth the risk of the occasional case of toxicity to iodine which may appear, however.

D'Antoni[6] has to date a total of 126 completely followed-up cases of amebiasis, treated with diodoquin, a course of treatment consisting of the administration of three tablets of diodoquin (3.2 grains each) three times a day for 20 days. One hundred and twenty-four of these patients were cured, though in five cases a second course of treatment was necessary. The minimum criterion of cure is three negative stool specimens secured at least two weeks after the completion of treatment and preferably spread over a week. Most of the cases in this series, however, were examined by much stricter criteria. The number of stools examined averaged more than five for each patient, and some patients were under observation for periods ranging from 12 to 18 months.

Two of the patients in which diodoquin failed were cured eventually by the use of combinations of amebicidal drugs. Various combinations were used with poor results. The plan was then conceived by administering chiniofon long enough to produce six to 10 diarrheic stools (about two days), for the mechanical cleansing effect of the diarrhea, and of following this medication by the administration of diodoquin in the usual dosage for five days. By this combined treatment one patient was cured in seven weeks, and since the conclusion of treatment has had 12 negative examinations, including two of material aspirated at sigmoidoscopy. The other patient was cured in eight weeks, and since has had 17 negative examinations including three of aspirated material.

Only four of these patients treated with diodoquin have exhibited mild toxic manifestations, chiefly headache and malaise, in only one instance enough to require bed rest for 24 hours. All of these patients had rather severe amebic infections and it is D'Antoni's belief that the toxemia was not caused by the drug but was due to the absorption of toxins from dead and disintegrating *Endamoeba histolytica*.

SULFASUXIDINE
(*Succinylsulfathiazole*)

Sulfasuxidine is one of the newer compounds in the field of sulfonamides. Because observations on this drug have shown that: (a) the flora of the gastrointestinal tract is profoundly altered by it; (b) it has definite bacteriostatic action in vivo; (c) absorption from the gastrointestinal tract is poor; (d) severe toxic reactions

have not been encountered; and (e) its effect on bacillary dysentery is marked. This drug has been used in the treatment of amebic dysentery.

The mode of action is not clear. It is doubtful that sulfasuxidine has any direct action against ameba *per se*. It may, however, be that the drug acts to modify the secondary bacterial flora, thereby aiding in the healing of the ulcers and so supporting the host that it may be able to overcome the amebic infection.

Knotts, et al.[6] presented six cases treated with this drug and their results were highly favorable. My main criticism on their series of cases is that only six cases were reported and their criteria of apparent cure of these cases were hardly what most investigators consider adequate enough.

I shall not attempt to give their method of treatment because at the present time we have more efficient drugs. Until more cases are reported on this drug and a stricter criteria for apparent cure is established, we are forced to consider this drug simply as an experimental drug as far as the treatment of intestinal amebiasis is concerned.

MISCELLANEOUS

Mention must be made of the bismuth salts, the nitrate and the less toxic subcarbonate which have a definite place in treatment. These bismuth compounds act in a purely mechanical manner as protectives and demulcents to the mucous membrane of the colon. They are indicated in all cases where amebic ulcers are slow to heal and where, as frequently is the case, secondary pyogenic infection has taken place.

CONTRAINDICATIONS TO COMMON AMEBICIDES

Emetine: Emetine should not be used if organic disease of the heart or kidneys is present. It is also contraindicated in children unless there is severe dysentery which has failed to respond to other measures. Emetine is best not used during pregnancy.

Chiniofon: Chiniofon should not be used in the presence of iodine intolerance and must be given with caution to patients with thyroid disease. Liver damage is also a contraindication. In those individuals in whom a persistent and severe diarrhea occurs from the drug, chiniofon should not be given.

Vioform: The contraindications for vioform are the same as for chiniofon.

Diodoquin: The contraindications for diodoquin are the same as for chiniofon and vioform.

Carbarsone: Carbarsone is contraindicated if renal or hepatic disease is present. The patient's vision and skin especially must be carefully observed for manifestation of arsenical poisoning.

THERAPY OF AMEBIASIS

The modern therapy of amebiasis includes much more than the treatment of that stage of the infection known as amebic dysentery, although in most works upon the subject treatment is practically confined to the treatment of acute and chronic amebic dysentery. The treatment of amebiasis includes the treatment of carriers, those having mild symptoms of infection, those presenting definite attacks of diarrhea, those developing acute and chronic amebic dysentery, as well as those having abscess of liver, lung and other organs or tissue.

The treatment of carriers is of tremendous importance at all times in the control of the disease and more so now that the American soldiers are returning from heavily infested areas from all over the world. In this infection we have the apparent paradox that the patient, when suffering from active symptoms, is relatively harmless so far as transmitting the infection is concerned, but becomes a danger when he has recovered from the infection. Cysts are the only form which is infectious because the motile forms are destroyed by gastric hydrochloric acid if ingested. The carrier remains a potential source of infection to himself and to others and is an extremely serious public health problem, especially if he handles food. Amebic abscesses of the liver, brain and lung may develop in carriers who have had little or no previous symptoms of intestinal amebiasis.

The criteria as to whether amebic infection of the intestinal tract has been cured

depends on laboratory and not medical examination. Disappearance of symptoms does not mean cure of amebiasis. Evidence of cure is acceptable if it consists of negative stool examination at weekly intervals for four weeks after completion of treatment, and at monthly intervals thereafter for five months. Repeated courses of medication are sometimes required to effect a cure. Treatment should be resumed whenever the stool again becomes positive for amoeba.

Amebiasis occurs throughout the United States, and the medical profession should abandon the idea generally held at the present time that amebiasis is a tropical disease.

The most important aspect of treatment is actually the control of this disease. The prophylaxis of amebiasis is that of any disease transmitted by contaminated food and drink. As mentioned previously the detection and treatment of carriers is the most important prophylactic measure. By and large, especially in places otherwise well sanitated, the food handler who is a carrier is the most significant factor in the transmission of the disease.

TREATMENT OF CARRIERS

In carriers a drug should be used which will not interfere with the individual's work and which can be taken without discomfort or injury to health. These requirements are fulfilled by several of the amebicides, namely: carbarsone, chiniofon, vioform or diodoquin. Chiniofon is usually the drug of choice, but diodoquin will probably take its place in the years to come. Chiniofon, three to four pills (4 grains each) three times a day for eight to 10 days may be given. The full dose sometimes has the disadvantage of causing severe diarrhea. It is not a toxic drug and may be used safely in mass treatment; the treatment does not interfere with the occupation of the patient unless it gives rise to severe diarrhea. A single course of treatment is usually curative in asymptomatic carriers, but the course may be repeated, if necessary, after an interval of two weeks has elapsed. No precautions are necessary as regards exercise or diet. If cysts are not eliminated with two courses, diodoquin, carbarsone or vioform can be tried.

Diodoquin, three to four tablets (3.2 grains each) three times a day for 20 days is given, but larger doses may be tried. Carbarsone, one capsule (4 grains each may be given twice a day for 10 days and repeated in 10 days if necessary. Vioform is administered in the form of one capsule (4 grains) three times a day for 10 days and repeated in 10 days if necessary. In very resistant cases a combination of chiniofon and diodoquin has been very efficient in D'Antoni's[6] series of cases. Chiniofon is given for two to four days, enough to produce six to 10 diarrheic stools, and then diodoquin in customary dosage is given for five days.

ACUTE DYSENTERY

Bed rest for patients with diarrhea is essential. Acute symptoms can be controlled with 1 grain of emetine hydrochloride given subcutaneously once a day until symptoms have subsided (which is usually four to six days) but not to exceed 12 grains. Diodoquin, chiniofon, carbarsone or vioform can be given once the acute symptoms have subsided. Emetine finds its most useful therapeutic field in the control of the acute dysenteric symptoms. Chiniofon, carbarsone and vioform can be used in the same dosage as mentioned for carriers. Diodoquin can be given in larger doses with better results—10 to 12 tablets three times a day for 20 days.

While the patient is being treated with emetine, a careful watch should be kept for symptoms of intolerance, as this drug is sometimes toxic even in therapeutic dosage. The symptoms of emetine poisoning are severe diarrhea, neuritis, myocarditis, great muscular weakness and nervous prostration; if any of these symptoms appear the drug should be discontinued.

Emetine-bismuth iodide may be substituted for emetine hydrochloride; it has the advantage of oral administration, and is thought by some to be more efficient. This drug is supplied in enteric coated pills (3 grains). Not more than 3 grains should be

administered per day, preferably at night, for twelve consecutive days. After completion of the treatment, the stools should be examined and if cysts or trophozoites are still present, chiniofon or any other of the amebicides should be administered as already recommended.

In D'Antoni's[6] experience, retention enemas of amebicidal drugs have never been necessary and he seriously doubts their rationale. "The introduction of amebicidal solutions, at most 200 c.c., into the lower bowel can scarcely be expected to correct an infection involving the colon, sometimes in its entirety." In view of the efficacy of present day amebicides, the indications for treatment by retention enemas seem very limited.

In the treatment of the dysenteric stage of amebiasis with any of the drugs mentioned the patient should remain in bed until the completion of the treatment in order to receive the best results.

CHRONIC AMEBIC DYSENTERY

The chronic form of amebic dysentery may follow severe acute attacks or may be chronic in type from the beginning, there being a history of repeated attacks of diarrhea in which small amounts of blood and mucous occurred in the stool.

If patients are seen during acute exacerbations with severe dysentery, the treatment should be that already described for acute amebic dysentery, while if the patient is seen during intervals between dysenteric attacks, a course of diodoquin or another of the previously mentioned amebicides is indicated.

In patients who have suffered from many relapses of acute amebic dysentery, the prospects of cure with any method of treatment are poor. Even if a cure of the infection is secured, the patient may continue to have attacks of diarrhea if extreme ulceration has occurred in the intestine resulting in the replacement of large areas of mucous membrane with scar tissue through which absorption of fluids is impossible.

Ghosh[7] suggests that in treating cases of chronic amebiasis attention should be paid to the adjustment of the diet in such a way as to keep the reaction of the stool alkaline. Carbodydrates tend to make the stool highly acid. Alkali by mouth has little influence on the reaction of stools. Increased protein diet and decreased carbodydrate invariably render the stool alkaline. Ghosh states that positive deductions can not be drawn from his three cases but that it is fairly possible that this point might help in cases which persistently do not improve with what seems to be adequate treatment.

The general treatment of amebiasis in those suffering from diarrheal or dysenteric attacks consists in bed rest and attention to the diet and general physical welfare of the patient. If dysenteric symptoms are very acute, it is best to withhold all foods for two or three days, and if food is given it should consist of barley water, albumin, milk with lime water and thin broths. When the acute symptoms subside, poached or soft boiled eggs, soft puddings and thick gruels may be given and as the patient improves a light soft diet may be substituted. Extreme care is necessary to avoid foods that are known to irritate the intestine. Alcohol should be forbidden to all individuals who have symptomatic amebiasis, and even in carriers excessive indulgence in alcohol often results in appearance of the symptoms of diarrhea or of dysentery.

AMEBIC HEPATITIS

The treatment of amebic hepatic infection is entirely surgical, although it should be realized that this may consist of either conservative or radical measures. Early recognition of amebic hepatitis and the institution of conservative measures during this presuppurative stage may prevent progression to actual abscess formation. Even in cases in which early abscess formation has occurred, conservative measures are frequently sufficient to effect complete resolution.

The term conservative therapy is used to signify the administration of the specific drug emetine hydrochloride, with or without aspiration, depending upon the presence of indications for the latter. In early cases

of amebic hepatitis, utilization of emetine alone may be sufficient. In other cases, however, a true abscess has apparently developed which requires, in addition to the emetine, evacuation of its content by aspiration.

At present, it is the concensus of most authorities that emetine hydrochloride is the most valuable drug in amebic hepatic involvement, and even large abscesses may completely disappear under emetine therapy alone. Every case of suspected amebic hepatitis or amebic abscess should be given the advantage of a course of emetine before any procedure is used, unless rupture of the abscess appears imminent. In many cases, especially in those with early small abscesses, no other therapy will be required. However, in the majority of cases in Ochsner's[8] series, evacuation of the abscess contents was necessary.

The evacuation of amebic abscess reigns totally in the realm of surgery and the technics used are beyond the scope of this paper.

The drug is preferably administered subcutaneously, as emetine hydrochloride in daily doses of 1 grain until 10 grains have been given. Because of its toxicity in excessive dosages and its cumulative action, the noxious effects of this drug should be realized and considerable care exercised in its use.

Immediately after completion of therapy of the amebic hepatic condition the patient should be given a course of therapy for the intestinal amebiasis. Even though the latter cannot be demonstrated clinically, it should be assumed to exist once the diagnosis of either amebic hepatitis and hepatic abscess has been established.

CONCLUSIONS

1. Emetine has no rival in the treatment of the acute symptoms of intestinal amebiasis and in amebic hepatitis, and even presuppurative amebic abscess.

2. On the basis of D'Antoni's 126 cases treated with diodoquin it is found that this new drug is essentially non-toxic in therapeutic doses, and is 92 per cent effective in single courses and 99 per cent effective if two or more courses are used in the treatment of intestional amebiasis. No drug previously used in the treatment of amebiasis has given such consistently satisfactory results. A word of caution, however, is necessary. This is a small series and one will have to observe more cases before the true evaluation of efficacy can be established. Diodoquin is apparently the most effective drug presently available for the treatment of amebiasis.

The claims for most of the amebicidal drugs were evaluated before the zinc sulfate technic for cysts and ova came into use. Evaluation of the amebicides using this technic as a criteria for cure should be done before a true knowledge of the efficacy of each drug can be accurately evaluated.

3. In the treatment of chronic and suppurative hepatic abscess, emetine plus surgical aspiration is the method of choice.

4. Other extra intestinal complications of amebiasis, such as lung abscess, are totally surgical procedures beyond the scope of this paper.

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THE EFFECT OF VITAMINS ON THE INTESTINAL FUNCTION

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NEW ORLEANS

INTRODUCTION

It was some time ago, when I was a premedical student, that I first became interested in vitamins. There were several reasons for this interest: (1) the usual interest shown by the laity and the professional groups over these new "energy pills"; (2) the fact that my family was more than the average "vitamin addict"; and (3) the observation of what seemed to be a "miracle" of treatment. This observation, viewed in the present light of knowledge concerning the function of vitamin B_1 could, I believe, truly be termed a miracle. The miracle had to do with a man, aged 33, diagnosed by the local physician as a case of low intestinal obstruction. The condition had existed for five days, and had been verified by a surgical consultant. For some unknown reason, the local physician decided to give the man a massive dose of vitamin B_1, intravenously, the day before the operation was scheduled. The condition cleared up in a matter of hours, and the patient was back on his feet and feeling fine in a week. The patient has remained well and in good health since. Whether this effect was due to B_1, whether the true diagnosis was missed, or whether something unforeseen and unrecognized happened is purely a matter of conjecture. Of course, you the reader, like me, will be tempted to say that the "cure" was certainly not due to the B_1 administration; but, in reality, who can say definitely yet.

Nevertheless, no matter what the effect of the vitamin B_1 on this patient, it did have the effect of stimulating interest on my part in the effect of vitamins on the intestinal function.

The present trend of study in this field has turned, more or less as a matter of necessity, to a study of the intestine in the various deficiency states and the effects of vitamin administration in these conditions. With the few exceptions that will be noted later, the administration of vitamins to the normal person has very little or no effect; and hence a study along this line would be much less fruitful than the above.

Vitamin deficiency states may be of two types: (1) primary, and (2) secondary. A *primary deficiency* is one which occurs without obvious cause. It is one in which, however, a consistently deficient diet may be a factor, such as in sprue or pellagra. A *secondary deficiency* is one which is caused by, or is associated with, a recognizable disease of the alimentary tract. It is seen, however, that the manifestations of the deficiency *per se* would be the same in each; although they may be partially or completely masked in the secondary type.

A state of vitamin deficiency, either clinical or pre-clinical, may develop due to inadequate intake. On the other hand the intake may be adequate but for various reasons, physiologic or pathologic, there may be increased need, decreased absorption, increased destruction, or decreased utilization of the vitamins. These factors may be present singly or in various combinations.

In a study of the small intestine in deficiency states, abnormalities may be recognizable incidentally during the usual gastrointestinal series and fluoroscopy. The best method for its study possibly, is repeated roentgen examinations at 30 minute intervals after the ingestion by the patient of a barium sulfate suspension on a fasting stomach. The films are usually developed and inspected at once, and at this time any needed modifications to the procedure may be made. Fluoroscopic examinations are also made during the study. If the $BaSO_4$ has entered the cecum, food is usually given at five hours, and the study is completed.

Obviously the abnormalities of the small intestine associated with vitamin deficiencies vary with the severity and duration of the disorder. Upon institution of therapy, the improvement in intestinal function usually lags behind the clinical improvement of the patient.

There are no special examinations for observation of the large intestine during deficiency states, although the barium enema, gastrointestinal series, proctoscopy, and stool examination are helpful procedures.

The various vitamins will be taken up for study in alphabetic order.

VITAMIN A

Vitamin A is fat soluble. It is an unsaturated alcohol derived from the reddish yellow pigment carotene. When taken in the food, about 20 per cent of this carotene is converted into vitamin A in the liver where it is also stored. Bile is necessary for the absorption of carotene, which is absorbed much less readily than vitamin A. Absorption of both, however, is favored by the presence of fat in the intestine.

Vitamin A is essential for the maintenance of the normal function and integrity of the epithelial tissues of the animal organism. As a result of deficiency of this essential substance there is atrophy of the epithelium concerned, conversion into a stratified squamous variety, and suppression of the normal secretions. As a result of these changes, the susceptibility of the tissues to intercurrent secondary infection is increased; for it is a well known fact that healthy mucous membranes with their protective secretions are the "first line of defense" against infection. However, the deficiency of just about any essential nutritional requirement will lower resistance to infection; and the physician is highly speculative who sees any beneficial results from vitamin A therapy during an acute infection or as a prophylactic measure, unless such a specific deficiency is present. Stryker and Janota[1] have made observations on the permeability of the intestinal wall in rats deficient in vitamin A and in control animals, using *S. enteritidis* and toxin of *Cl. botulinum* as test substances. No noteworthy differences between the two groups of rats were demonstrated. This work has been confirmed by others.

Hyperkeratosis with ulceration of the forestomach of rats as a result of vitamin A deficiency has been repeatedly reported. However, most authorities are of the opinion that this cannot at all be regarded as a specific lesion.

The most likely pathologic changes in the gastrointestinal tract in vitamin A deficiency are alterations in the teeth, inflammatory or atrophic changes in the oral, stomach and intestinal mucosa and inflammatory or cystic changes of the accessory glandular organs. These changes may interfere with absorption from the intestine and may lead to hypochlorhydria or achlorhydria. Symptomatically the most common complaint, and the only gastrointestinal symptom which has been noted with any frequency, is diarrhea. The cause for this diarrhea is not clear, although it seems most likely to be related to atrophic or inflammatory changes in the mucosa of the large or small intestine or perhaps to the achlorhydria. However, at autopsy, the gastrointestinal tract is usually comparatively normal. This paucity of gastrointestinal symptoms and pathologic changes in man is surprising when one recalls that a large part of the gastrointestinal tract is made up of epithelial tissue.

Daily requirements of vitamin A are 3000 units for the normal adult, 6000-8000 units for the growing child, and 5000 units for the pregnant or nursing woman. The requirements are, of course, increased in a specific deficiency state in proportion to the severity of the condition. The experimental basis for the use of vitamin A in gastrointestinal disorders is not yet clearly established.

Hemorrhagic diarrhea may be part of a toxic syndrome caused by over dosage.

VITAMIN B (THE "B" COMPLEX)

This group of vitamins which are grouped under the heading of vitamin B are water soluble and are stored very poorly in the human body.

The following findings have been noted in numerous cases studied extensively by various authorities in which some deficiency condition appeared to play either a major or minor part. The patients were diagnosed as having a vitamin B deficiency because of their response to specific therapy:

A. Motility

1. Hypermotility: In the earlier, less advanced stages rapid passage of the barium sulfate was repeatedly noted.

2. Hypertonicity: This finding like the above was noted in the earlier, less advanced stages. It was often evidenced by a reduction in the size of the lumen to one-half or even one-quarter of its normal size.

3. Hypomotility: In the later stages of the deficiency state, slow passage of barium sulfate was noted. This, with the hypotonicity often results in dilatation of the colon with degeneration of the mucous membrane.

4. Hypotonicity: This also was noted in the later stages of the deficiency state, and was evidenced by a dilatation of the bowel.

5. Abnormal segmentation: Areas of spasm of variable length interspersed with areas filled with barium sulfate served to give the picture of numerous discrete masses of barium. In advanced stages, such as in non-tropical sprue, for example, these filled segments are usually associated with hypomotility. In the earlier stages, the masses of barium sulfate are shorter and may be the width of the normal lumen or even narrower; this is called hypertonic segmentation.

On fluoroscopic examination little forward progression of the opaque media is seen; however, there is an almost continuous activity, with jumping of the areas of contraction back and forth. Pressure on a distended segment often results in an immediate contraction and a shifting of the opaque material.

6. Scattering effect: In some cases, as the main mass of barium sulfate passes on, small irregular masses linger behind and give small shadows of irregular size and shape. This effect seems to be due in part to a disturbance in the function of the circular muscle and in part to a disturbance in the movement of the mucosa which depends on the muscularis musoca.

7. Gas and fluid levels: In the advanced cases this finding may be marked, and flat plates of the abdomen may suggest the possibility of ileus. The presence of gas in considerable amounts is thought to be a manifestation of a disturbance in the ability of the mucosa to absorb gas.

Whether these findings are due to a primary defect of the intrinsic nervous mechanism, or to a secondary one, due perhaps to a vitamin B deficiency, has not yet been completely settled; but there are very strong arguments in favor of the latter.

B. Mucous Membrane

1. Reduction to complete obliteration of the mucosal folds may be seen, depending upon the stage of the condition.

2. Exaggeration of the mucosal folds, on the other hand, may be seen. The cause of these conflicting findings is not well understood.

C. Gastric Retention

This is frequently found in well marked primary deficiency states; and a good sized six hour residue of barium sulfate may be present, with sluggish, ineffective peristalsis and often with antral spasm.

D. Disturbance in Carbohydrate and Fat Digestion and Absorption

In the hypomotile type of chronic enteritis, evidence of impaired fat digestion and absorption is found, just as it is in the hypermotile type. This finding may be explained in the hypermotile type by the rapid passage through the small bowel; but obviously this could not be the explanation in the hypomotile type.

Free and Leonards[2,3] have shown that, in rats deficient in vitamin B, the intestinal absorption of galactose was 35 per cent less than in normal rats. Later work showed the rate of intestinal absorption of galactose to be impaired 66 per cent by thiamin deficiency, 12 per cent by pyridoxin deficiency, 15 per cent by pantothenic acid deficiency, and unaffected by riboflavin.

E. Miscellaneous

1. Dalldorf, Gilbert and Kellogg[5] showed that gastric ulcers appeared in a large percentage of rats fed a B_1 deficient diet, and therefore suggested that B_1 therapy should be instituted in the treatment of gastric ulcers in humans. This work has been confirmed by others. However, most authorities are of the opinion that the ulcers arise secondarily in the course of the vitamin B_1 deficiency, probably as part of the response of the stomach to a generalized systemic deficiency condition.

2. It has been found that "folic acid" *and biotin promote the synthesis by residual intestinal bacteria[6] of additional essential dietary factors. Thus these substances may prove to be a valuable adjunct to sulfonamide therapy, as this drug affects primarily the vitamin synthesizers (colon bacilli) and leaves more or less uninfluenced the vitamin requirers (lactobacilli, streptococci, and anaerobic bacilli). Para-amino-benzoic acid partially counteracts the effect of the sulfonamides, especially the chief offender, sulfasuxidine, on vitamin synthesis in the intestinal tract.

A large number of gastrointestinal symptoms, including anorexia, constipation, diarrhea, dysphagia, flatulence, and vomiting, have been ascribed to deficiency of components of the B complex. Of these symptoms it is clearly established that anorexia and diarrhea accompany vitamin B_1 deficiency in man.

It is quite clear that thiamin and nicotinic acid are essential for normal functioning of the gastrointestinal tract. It is difficult to establish a definite part of the B complex as the specific agent in the etiology of the various and sundry changes in the gastrointestinal tract. It is much easier to ascribe the changes to the group as a whole. It is undoubted though, in an over all view, that B_1 is by far the most important member of the group, with nicotinic acid as very close second.

Cheney[7] treated three groups of patients with vitamin B_1 with the following results:

1. Chronic diarrhea: Seven patients with chronic diarrhea of unknown etiology were treated with thiamin and three of them also received liver extract. Five of them were asymptomatic except for their diarrhea. All seven patients responded to the therapy.

2. Mucous colitis: This group was comprised of six patients, all of whom received vitamin B_1 and all but one of them received liver extract during their course of treatment; all improved with treatment.

3. Idiopathic ulcerative colitis: There were 11 cases in this group, and all but one was definitely improved while taking injections of liver extract with vitamin B_1.

Chesley, Dunbar, and Crandall treated 44 patients with the so-called functional gastrointestinal disturbances. Such disturbances perhaps constitute the majority of all digestive conditions for which the physician is consulted, and are ascribed by a great many authorities to vitamin deficiencies. Their results with vitamin B complex and nicotinic acid are as follows[8]:

	Per cent satisfactory in vitamin B complex	Per cent satisfactory in nicotinic acid
Flatulence	67	57
Abdominal distress	64	56
Alternating constipation and diarrhea	74	69
Constipation only	75	71
Diarrhea only	50	50
Weakness and fatigue	60	17
Nervousness	63	16
Anorexia	100	30

Williams, Mason, Wilder, and Smith conducted a study to determine what happens when the diet is markedly deficient in thiamin. Extreme anorexia, nausea, vomiting, and constipation were prominent in the train of symptoms. The earliest effect of vitamin B_1 deficiency is loss of appetite. This occurs some time before the appearance of polyneuritis; it is undoubtedly due in part at least, to the atony of the gastroin-

*The term "folic acid" refers to concentrates which furnish an essential growth factor for *L. casei*. Although the activity of such a concentrate is ascribed to the "folic acid" content, other substances are present, and in reality only a small amount of folic acid.

testinal tract. Thiamin is undoubtedly the one vitamin most likely to be deficient in the average American diet. This is a good theoretical explanation for the etiology of the large number of people who are so "finicky" about their food.

The daily requirement of thiamin in the normal individual is 1.5 to 2.3 mg. as established by the Food and Nutrition Board of the National Research Council. This requirement is, however, influenced by several factors, such as the basal metabolism, the quality and the quantity of the diet, periods of active growth, pregnancy, and lactation. It is increased by a diet high in carbohydrate but decreased by one high in fat.

Vitamin B_2, at first designated as a part of the B complex, has since been shown to be itself a complex—in the B complex. It contains several factors, the most important of which are riboflavin, nicotinic acid, pyridoxin, and pantothenic acid. There are various other members of the group which at the present time are thought to be unimportant.

Riboflavin, *per se*, has little or no demonstrable effect on the gastrointestinal tract. Its daily requirements are 2.2-3.3 mg.

Nicotinic acid (niacin) is the well known P-P (pellagra preventing) factor in the B complex. One of the most outstanding features of pellagra, the result of a pronounced deficiency of this factor, is anorexia, digestive disturbances, and diarrhea, which may occasionally prove intractible. There is sometimes constipation. In every case of pellagra in which nicotinic acid has failed to relieve the diarrhea, there is some organic lesion to account for this failure usually. The daily requirements for a normal individual are 15-20 mg. In pellagra the dosage varies with the physician, but a good program is as follows: 1,000 mg. intravenously on the first day, and 500 mg. intravenously daily for a week, followed by repeated smaller oral doses. This dosage is extremely variable, however.

Pyridoxin (vitamin B_6) has little known effect on the digestive tract. Some work has been done recently concerning the use of this factor in the treatment of "morning sickness." The daily requirement for the normal person is about 2 mg.

Pantothenic acid is also still rather obscure as concerns its effects. Intussusception has been observed in rats with pantothenic acid deficiency, the significance of which remains unknown. The daily requirement for the normal individual is about 5 mg.

VITAMIN C (ASCORBIC ACID; ANTI-SCORBUTIC; CEVITAMIC ACID)

Vitamin C is water soluble. Its essential function is to maintain the integrity of the intercellular ground substance in which tissue cells are distributed. In scurvy, the effect of vitamin C deficiency, there is, as indicated above, a reduction of the intercellular cement substance, resulting in a weakening of the endothelial wall of the capillaries. The outstanding gastrointestinal symptoms in scurvy are related to gingivitis. Anorexia and diarrhea may be present. Study of human material does not support the new theory that the gastrointestinal tract suffers in a conspicious fashion from vitamin C deficiency; however, minute hemorrhages and occasionally larger ones with secondary necrosis and ulceration are found.

A great deal of controversy has surrounded the possible role of vitamin C deficiency in cases of peptic ulcer, other ulcerative lesions of the gastrointestinal tract, and in bleeding from ulcers and other lesions. The urinary excretion of vitamin C is large in patients with ulcers, and the capillary fragility test will often show that vitamin C has failed to be absorbed and stored, but this may be due to a decreased intake or absorption, or increased destruction. The relation of hemorrhage from the gastrointestinal tract due to vitamin C deficiency remains unsettled. It would seem that since vitamin C certainly is not harmful and might possibly be helpful, that it should be an addition to the diet of patients with gastric ulcer.

There have been numerous clinical reports, especially from European workers, that vitamin C occasionally gives rise to in-

testinal colic and to an increase in intestinal motility. Haag and Taliaferro[9] have observed the effect of ascorbic acid on the isolated guinea pig colon. They found that ascorbic acid in concentrations ranging from 1-10 mg. per cent caused a great increase in tone in from one to two minutes after application. Thereafter the tone gradually decreased until it reached normal.

The daily requirement of vitamin C for the normal adult is 75-100 mg., but this varies considerably, being increased in infections and other conditions which tend to deplete the vitamin stores. Such an intake is absolutely indicated in all cases of gastrointestinal ulceration. In acute deficiencies one may give as high as 1000 mg. for a few days and then 100-300 mg. daily until the deficiency state is under control.

A substance similar to vitamin C and having practically the same effects has been reported by Russnyak and Szent-Gyorgyi and termed "citrin." Its existence is now disputed by C. S. King and others.

VITAMIN D

This is a fat soluble vitamin, a deficiency of which causes rickets in the child.

In animals there is very little, if any, evidence that deficiency of vitamin D leads to impairment of gastrointestinal function or structure. Atony of the intestine has been described as an accompaniment of rickets, but the relation of it to deficiency of vitamin D is not clear.

In childhood, when there is defective absorption of fat and calcium, a fatty diarrhea may develop as part of the syndrome characterized celiac rickets. The absorption of vitamin D is apparently defective in this condition; and it responds to the administration of the vitamin, as does a similar condition in adults called idiopathic steatorrhea or non-tropical sprue.

Daily requirements of vitamin D are 400-800 units daily, and possibly a little more during pregnancy and lactation. Experiments are under way using massive parenteral doses of vitamin D.

Some adults who are treated with large doses of vitamin D, for some reason may develop intense diarrhea along with other symptoms of overdosage.

VITAMIN E (ANTI-STERILITY)

This vitamin E is a fat soluble vitamin. Evidence is lacking that a deficiency results in significant gastrointestinal abnormality.

VITAMIN K (ANTI-HEMORRHAGIC)

This is a fat soluble vitamin. Bile is necessary for its absorption, and fat interferes with its absorption.

Evidence is not complete enough to suggest that deficiency of vitamin K may result in any abnormality of gastrointestinal function or structures other than those changes associated with bleeding. Briefly, it is of value in the treatment of the hemorrhagic diseases caused by a deficiency of prothrombin, such as hemorrhagic disease of the newborn and hemorrhagic diseases associated with certain diseases of the gastrointestinal and bilary tracts. Dosage is estimated and determined by a study of the prothrombin level. Toxic vomiting and diarrhea may result from overdosage.

SUMMARY

In the above discussion, a number of important actions of the various vitamins on the intestine and the intestinal function have been described and discussed. While some of these effects are clearly established, others are not; and, as a consequence, many of the findings enumerated are not universally agreed upon. An attempt was made to stress the established findings and indicate the others. This was done by means of the presentation of both laboratory and clinical data.

In the diagnosis of vitamin deficiency states such as have been studied, it is curious that evidence of deficiency of most of the vitamins is to be found in the mouth. This fact should be another stimulus to the modern practitioner to return somewhat to the time-taking physical diagnostic methods of his predecessors rather than rely too much upon the mechanical contrivances now at his disposal.

The physician should also remember that vitamin deficiencies are usually multiple, and hence not expect to find the typical pic-

ture of a single vitamin deficiency. He should remember this when he thinks of treatment; but first of all he should never forget that there is no completely satisfactory substitute for an adequate diet.

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FILARIASIS

A FUTURE PROBLEM IN THE UNITED STATES

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SHREVEPORT

With the entrance of the United States into the present world conflict in 1941, tropical diseases and tropical medicine gained a position of importance which they have never before occupied in this country. This was the result of the global war which placed hundreds of thousands of American service men in tropical and subtropical countries where these diseases are found in their most severe forms. And now, after three years of war, the medical profession has been duly impressed with this new importance of tropical medicine: not only those doctors who are in the battle areas who, of course, were the first impressed but also the physicians here at home. No longer are most tropical diseases remote entities in textbooks, for daily, soldiers, sailors, and marines are returning to this country with some form of leishmaniasis, trypanosomiasis, schistosomiasis, malaria or filarisis.

It is the purpose of this paper, then, to present a discussion of one of these timely diseases—filariasis—in the light of some of the newer concepts which have been formulated since the beginning of the war, and more specifically to consider the possibility and probability of filariasis as a post-war problem in this country.

There is, perhaps, no tropical disease which is feared more by the general public and the soldier than filariasis. This is because the only familiar stage of the disease is the chronic one presenting a picture of an enlarged scrotum or elephantoid extremities. It is no wonder, then, that the services turned to the medical profession with urgent questions concerning the prognosis for life, for sexual impotence, and for permanent disability if they contracted the disease. But while all these questions were being considered, an even more significant question that lurked in the minds of many was the possibility of filariasis becoming endemic in the United States. They had not forgotten that just 100 years ago there was an endemic focus of filariasis at Charleston, South Carolina.

If then, there are still potentially endemic areas for the disease in this country, it is not difficult to imagine the problem that might be created with new cases of filariasis returning to this country each week from the Pacific area. The real scope of this problem can only be ascertained after we know the number of cases of filariasis which have returned to the United States. Unfortunately, this fact is not available at the present time but conservative estimates place the figure at about 7,000 men.

With this problem before us, we will consider filariasis in some detail in an effort to get an insight into a situation which theoretically could produce a tremendous hazard to the health of the American public.

GENERAL CONSIDERATIONS

Under the general term filariasis, there are included all the morbid conditions pro-

duced by certain parasitic nematodes of the superfamily *Filarioidea*, the adults of which may live in the circulatory or lymphatic systems, the connective tissues, or serous cavities, while certain larval forms, called "microfilariae," are found in the circulating blood or in the lymph spaces. In its broadest sense, the term filariasis implies infestation of the host with any species of the superfamily and thus, widely different pathologic conditions may be included under it. Although more than twenty species of filariae have been reported for man, only four are the etiologic agents of important pathogenic types of filariasis. These agents, the diseases which they produce and the vectors are listed below:

Etiologic Agent	Disease	Vectors
1. Wuchereria bancrofti	Bancroft's filariasis	Mosquitoes (Culex, Aedes)
2. Wuchereria malayi	Malayan filariasis	Mosquitoes (Anopheles, Mansonioides)
3. Onchocerca volvulus	Onchocerciasis	Black Gnat (Simulium)
4. Loa loa	Loaiasis	Mango Fly (Chrysops)

Of these pathogenic types of filariasis, the first, Bancroft's filariasis is the most widely distributed and the most important clinically. Since all the cases of filariasis reported in this war among American service men are thought to fall in this category, the discussion will be limited to the one specific condition caused by *Wuchereria bancrofti*.

GEOGRAPHIC DISTRIBUTION

Bancroft's filariasis is found throughout practically all of the warm regions of the world. In Europe, it seems to be confined to Barcelona, Hungary and Turkey, while in Africa it is seen throughout the central tropical belt and along the northern coast. It is found throughout Madagascar, around the Arabian coast and has extensive distribution in Burma and India. In the Far East, it is especially prevalent in South China, extending as far north as the Shantung Province, southern Korea and southern Japan, and southwardly to the Dutch East Indies and other islands of the Pacific where in some localities, 80 per cent of the inhabitants are known to be infected. This condition is reported as far south as Brisbane in Australia. Although, the disease is recorded as widespread in Asia and the South Pacific, Craig and Faust[7] think that some of these records include or refer to *W. malayi* which was first specifically distinguished in 1927.

Since a large number of the cases seen among the American service men have come from Samoa, it is of interest to note that Hargrave reported that filariasis in American Samoa probably causes greater damage than any other disease by reason of the disability effect and the undermining of the general health which may predispose to other infections. Also, Phelps reported filariasis as the third cause of death in frequency in American Samoa, the first cause being tuberculosis and the second pneumonia. A survey of the native guard and civil employees there showed 48 per cent harboring microfilariae in their blood.

In the Western World, filariasis is common along the coast of northern South America, but apparently does not extend far inland in the Guianas, Colombia, or Panama. It is common in the Greater and Lesser Antilles. In the United States, Charleston, South Carolina, was formerly an endemic focus of infection. The filariasis cases were authenticated by early workers but in 1915, Johnson found that 19 per cent of the patients admitted to the Roper Hospital in Charleston harbored microfilariae in their blood and in 1919 Francis,[16] of the United States Public Health Service, found among 400 individuals living in Charleston, that 77 were infected with microfilariae bancrofti. Today, this focus has disappeared and no new cases have been reported. South Carolina is not the only part of the United States, however, where filariasis has been reported in the past for Matas, in 1913, reported upon the occurrence and treatment of elephantiasis in New Orleans and a certain number of cases have been reported among Puerto Ricans and Jamaicans in New York City.

ETIOLOGY

In all cases of filariasis reported among military personnel, the etiologic agent has been *W. bancrofti*, and *W. malayi* has not

been identified in any of the patients although its lesions may be identical with those of Bancroft's filariasis. For this reason, this discussion will be limited to *W. bancrofti.*

The adult male of this species measures approximately 40 by 0.1 mm. and the female about 90 by 0.28 mm. They are threadlike, creamy white and translucent and while the head is slightly bulbous, their terminations are bluntly rounded. The vulva in the female is cervical in position and the uterus, which extends throughout the greater portion of the body, contains the ova. These ova are thin ovoidal shells 38 by 25 microns in size and they contain the young embryos. As they are pushed into the outer portion of the uterine tubes, these shells become elongated to accommodate the uncoiling embryos and become known as the sheaths of the embryos. After exit from the parent worm in this form, the embryos are known as microfilariae and generally migrate to the blood stream via the lymphatics. They measure 127 by 320 microns in length by 7.5 by 10 microns in diameter and exhibit considerable motility. They can be readily seen in preparations stained with hematoxylin or Giemsa's solution.

In certain autochthonous infections especially in China, India, Australia and the Western hemisphere, the microfilariae exhibit a striking phenomenon referred to as filarial periodicity. This was first noted by Manson in 1880 when he observed that the larvae are found in the peripheral circulation in great numbers during the night, reaching a maximum between 10 p. m. and 2 a. m., although few or none can be found during the day. On the other hand, even prior to the present war, it was known that in the Philippines, Fiji, Samoa, Takelou, Wallis and Ellice Islands, where the morphology of the parasite is identical, native cases manifest no nocturnal periodicity. This has been borne out by Michael[23] and others in Samoa while studying the native population as well as the American service men with filarial manifestations. There have been numerous attempts to explain this periodicity and it has been suggested that it depends upon the sleeping habits and activity of the definitive host; or that it is related to the habits of the insect host; or that it depends upon a daily cyclic parturition of the female filariae with daily destruction of all the microfilariae. Since these theories cannot be considered here in any great detail, suffice to say that the phenomenon has not been explained with full satisfaction.

MODE OF TRANSMISSION

The parasite causing filariasis requires an intermediate host for development through the larval stages. This intermediate host then transmits the larvae to man. In the case of *W. bancrofti,* the proved intermediate hosts include 32 species of mosquitoes and undoubtedly other appropriate hosts exist. According to Mumford and Mohr[24] the chief vector of the microfilariae with nocturnal periodicity is the Culex quinquefasciatus while the carrier of nonperiodic microfilariae is usually Aedes variegatus. Other common vectors include Culex pipiens, Aedes rossi, and Anopheles costalis. The incrimination of these mosquitoes as the carriers of microfilariae in the Pacific island areas has been confirmed by Michael, Foegel, and Huntington, and others who have studied the disease among military personnel in these battle areas. The mosquito most commonly involved in Western Hemisphere filariasis is the Culex and extensive control procedures are responsible for decreasing the filarial incidence in this part of the world, especially in Brazil.

The microfilariae pass into the stomach of the insect with the blood meal and lose their sheaths in the first two to six hours. Some of them migrate through the wall of the proventriculus and cardiac portion of the midgut and in four to seventeen hours have reached the thoracic muscles. During the course of the next five to seven days, the microfilariae pass through two ecdyses and mature filiform larvae emerge which measure 1.4 to 2 mm. by 18 to 23 microns. This stage is usually completed about the tenth or eleventh day when the larvae migrate through the hemacele within the la-

bium of the proboscis. They escape through the tip of the proboscis most frequently during its flexure at the time the blood meal is being drawn and they enter the skin by active penetration either through the puncture wound or unbroken skin.

From this point of entrance, the infective stage larvae pass through the subcutaneous tissues and through the peripheral blood vessels to the lymphatics and lymph nodes. Here the larvae settle down and mature, mate and the females parturate. It is known that after a successful inoculation of man, the microfilariae, discharged by recently matured females, may be expected to appear in the peripheral blood in about twelve months. Therefore, the incubation period of the disease was always given at about twelve months because the mature worms were the only ones thought to produce symptoms. This concept has been somewhat changed in the light of our present knowledge because it has been shown among the American service men in the Pacific area that the incubation period and symptom free periods may be concomitant and shortened to as little as two months. This is explained by the fact that larvae probably neither reach the site of maturity nor become mature before they produce symptoms. Indeed, it is thought that many never become mature. The immature worms gradually accumulate in large lymphatic vessels or a set of lymph nodes and from this site, they may initiate symptoms.

PATHOLOGY AND PATHOGENESIS

There are many instances in which the presence of adult filarial parasites in the body is recognized by the occurrence of microfilariae in the blood while the parasites exercise no recognizable injurious influences or pathologic changes. On the other hand, many cases of pronounced pathology are seen. O'Connor and Hulse[28] have shown that marked pathologic changes associated with filarial infection may occur in some patients who have never complained of symptoms and are unaware of any disturbance.

It has been stated that there is a negligible skin reaction at the site where the mosquito inoculates the infective stage larvae. Napier,[26] however, points out that the skin around where the larvae penetrate may become thickened, hard and red, and this condition usually persists for some days. There is an inflammatory reaction set up in the lymphatic channels through which the larvae migrate apparently as a result of some substance secreted by the larvae, and the tissues respond by hypertrophy of the endothelial cells of the vessel walls.

After considerable study of the cases of acute filariasis among the American troops, Michael[23] made specific histo-pathologic observations. He found that with a living worm in the lymphatic channel, fibrin is deposited on the endothelial surface, the wall of the lymph vessel becomes edematous and markedly thickened and there is a heavy cellular infiltration of eosinophils. Degeneration follows and the tissue reaction becomes more specific. Worms ranging from partial degeneration to dead forms may be seen within the lumen and in the lymph channel wall. The vessels show a thickening by proliferation of filarial granulation tissue which is almost pathognomonic for the condition. Caseating foci of worm segment are represented by a central core of necrotic tissue with stellate radiating proliferations of endothelial cells, fibroblasts, epithelial cells, and numerous foreign body giant cells. There is a dense zone of eosinophils surrounding the area. The granulation tissue is arranged in palisades following wavy undulating pattern. This distribution conforms to the coiled segments of the parasite. It was noted that the calcification observed in degenerated worms started centrally and proceeded peripherally. The non-specific character of the granulation tissue has led Michael[23] and others to believe that the lymphangitis is due to a specific allergic reaction in response to the worm or microfilariae and a partial obstruction of the lymphatics.

A different histologic picture was described for the lymph nodes involved in this condition. In the nodes where the parasite was living and mature, the histologic pic-

ture was distinctive. Worms were present in the afferent lymphatics or the medullary sinuses. In most instances, afferent vessels were hyperplastic, forming varices which extended into the deeper portions of the lymph nodes. Growing worms held within these varices were surrounded by a rather specific type of tissue reaction. A dense zone of eosinophils surrounded the worm and a central focus merged into the edematous lymph follicles with intact germinal centers showing increased mitotic figures. The plasma cells were increased in cortical areas and the entire node was edematous. The enlargement of the node was believed to be due to the presence of the parasite, edema and the generalized hyperplasia of the node. With degenerative changes in the worm both the parasite and the lymphatic tissue of the host show histologic variation. The final outcome is one of dissolution and absorption of the parasite, fibrous tissue replacement and disappearance of most of the specific diagnostic criteria.

The adult filarial worms which degenerate and die and are encapsulated within the lymphatic tissues are thought to discharge toxic by-products which are in turn probably responsible for the acute inflammatory processes which usually develop around them. These reactions are presumably allergic in type and may be subclinical or with mild inflammatory manifestations. In support of this allergic hypothesis Acton and Rao[2] reported a series of cases with urticaria and eosinophilia believed to be due to filarial infection. This allergic reaction has been recently restudied by Michael[23] among the filarial cases in the American troops and he thinks that in the development of the classical pathologic picture of filarial involvement of the genitals, with later destruction, necrosis and even calcification, there is at first a stage of tissue sensitivity, manifested by edema and perilymphatic cellular invasion, due to the presence of the worm elsewhere.

It has been pointed out by Napier[26] that the most likely explanation for the periodicity of the febrile attacks and other allergic signs and symptoms, both local and general, is that the gravid female gives birth to living embryos intermittently, probably a few days each month.

The pathologic processes which are responsible for clinical manifestations of filariasis are inflammation and obstruction of the lymph channels. The inflammatory processes have been described in detail but the more serious aspects of this condition are due to some form of obstruction. The circulation of lymph in any part may be obstructed by single or bunches of adult worms in the lymphatic vessels, large or small. The blockage may be associated with little organic reaction or an inflammatory one may occur such as described above. If obstruction due to the adult parasites takes place as high up as the thoracic duct, large varicose dilations of the thoracic and retroperitoneal lymphatics may be produced. A similar obstruction of abdominal lymphatics may cause a chyluria if transudation of chyle through the distended or ruptured lymphatics into the pelvis of the kidney, the ureter, or the bladder occurs. In a like manner, varicose dilations of the lymphatic vessels of the inguinal, iliac, testicular, spermatic regions, and of the skin of the labia or scrotum may occur. Hydrocele may also result from filarial infection.

When the lymph flow is obstructed, the lymph pressure increases, the lymph ceases to drain from the tissues and the part becomes progressively more swollen. In the course of time, the fibroblasts in the skin multiply and form new fibrous tissue which makes the skin dense and hard; the deeper skin layers involving the sweat glands, interfering with the lymphatics in that region and producing edema followed by fibrosis around the sweat glands, which are eventually destroyed so that the skin in elephantiasis is harsh and dry. In the meantime, surface hypertrophy of the epidermis becomes more marked, fissures occur in the horny layer and allow microorganisms to invade the corium. In these large elephantoid limbs, repeated attacks of inflammations, originating at the surface and due to the secondary bacterial infec-

tion, are externally common and increase the local hypertrophy.

As long as the lymphatic obstruction is only partial or intermittent, microfiliariae will find their way into the blood stream, but if it is complete, the larvae are confined behind the obstruction in the edematous and hypertrophic limb and do not appear in the blood stream. Hence, it is the rule that in cases of chyluria or lymphatic varix of the cord, microfilariae are almost always found in the blood, whereas in elephantiasis of the limbs and genitalia they are frequently not found.

The importance of secondary bacterial infection is a controversial subject. It was thought by Anderson[3] and Grace[18] that the lymphatics in filaria-infected individuals was the result of hypersensitiveness to certain strains of hemolytic streptococcus. This belief came about when it was noticed that in elephantoid limbs, the skin became stretched and poorly nourished and with consequent cracking, the invasion by bacteria and fungi was comparatively simple. The controversy which arises is whether the micro-organisms isolated from the outer cutaneous layers of these limbs could be responsible for the preceding lymphangitis. Leiper, Acton and Rao are among the workers who agree with the importance of staphylococcal and streptococcal infections in filarial attacks. McKinley is opposed to this concept because he found no evidence of bacteria in the actual focal centers of the inflammatory processes. O'Connor[27] is one of the leaders of the school which believes that most of the mild inflammatory reactions can be attributed to the irritation of the filarial secretions and of the body itself and to an allergic response on the part of the host to these. In regard to this matter, it is of interest to note that comparatively few cases of streptococcal contamination have been encountered in the cases among the American service men in the South Pacific.

SYMPTOMATOLOGY

The clinical picture of filariasis can best be divided into four stages. These are: (1) the incubation period; (2) the patent symptomless period; (3) the acute stage; and (4) the chronic stage.

The incubation period is given in most textbooks as twelve months. This includes the time from the entry of the third stage infective larvae into the skin until microfilariae first appear in the peripheral blood. During this time, there are no known symptoms, except for occasional allergic manifestations. The second or patent symptomless period may last for years or throughout life and although local tissue alterations around the adult worms may be in progress, there are typically no manifest symptoms. Actually, in the light of recent studies by Michael and others, it is now believed that the incubation period and symptom free period may be concomitant and symptoms have been observed in as short a time as two months after initial exposure.

The occasional allergic reactions which are the only symptoms or signs seen during the initial two stages of the disease have been described by O'Connor et al.[27] They are generally thought to be due to the by-products of the migrating worms and they may cause the so-called "fugitive" swellings or lymphangitis. In spite of the fact that the incubation period may be as short as two months, it is to be remembered that a large number of cases will show an essentially symptomless period of months and years.

The acute stage is the one in which lymphangitis is the conspicuous symptom. It is characteristically recurrent and is usually seen with so-called "filarial fever." The lesion is often linear, elevated, hyperemic and excruciatingly painful to the touch. Constitutional symptoms not uncommonly usher in the attack and these include chills, mild fever, general malaise, headache, occasional photophobia, and pains all over the body. The painful, red swollen areas in the extremities later involve the adjacent lymph nodes followed by retrograde or centrifugal lymphangitis. The majority of the patients show funiculitis with or without epididymitis, orchitis, or hydrocele. Grace,[17] in his studies of tropical lymphangitis in New Guinea, states that

four-fifths of the patients show involvement of the lower limbs, followed by the upper limbs, breasts and scrotum. But in Michael's series as well as that of Buxton, and Dickson, Huntington and Eichold,[10] highest incidence showed involvement of the scrotum followed by the upper extremities and then the lower extremities.

This initial attack lasts for a few days to weeks and is followed by remissions. Michael reported that the majority of his cases had one or more relapses since the initial attack. During these remissions, the involved areas may regress and the patients show few, if any, physical signs. They may actually enjoy good health during this time and the periods of remission seem to grow longer and the duration of relapse shorter with each subsequent attack.

It is important to remember that it is this acute stage of recurrent lymphangitis which has been repeatedly observed among our service men in the Pacific isles. This is what the natives call "mumu" and it was first described by Buxton[6] over 20 years ago.

Another part of the symptomatology of this disease about which little is said is that of the psychosomatic manifestations which have not been uncommon among our troops. The apprehension of the soldiers and marines concerning this disease has been quite noticeable and it is attributed to their seeing elephantiasis in its worst form among the natives and the natural regard which they have concerning their genitals, and the possibility of becoming sterile. This apprehension has led to mental depression in some cases and the possibility of these manifestations must be remembered by the physician and guarded against.

It is the chronic stage of the disease about which one usually thinks when the term filariasis is used and although the acute stage has been long recognized, the majority of the literature is concerned with terminal filariasis or elephantiasis. When the chronic stage of the condition develops, there is enlargement of the involved organ or member in an elephantoid type of disease or the development of lymphocele, frequently with rupture, in the less fibrosed type. Elephantoid scrota and extremities may become tremendously enlarged and a great burden to the patient. The involved tissue usually consists of lymph and fat in a fibrous matrix covered by a stretched, tightened skin. The edema present is nonpitting in type.

The statistics of Manson-Bahr show that in 95 per cent of the cases, the lower extremities are the seat of the disease. It is usually, but not always, confined to below the knee. Next to the leg, the scrotum is most frequently involved and these tumors may become enormous. Commonly they are 10 to 15 pounds in weight but the largest one recorded is 224 pounds.

Elephantiasis usually begins as a lymphangitis with fever, secondary dermatitis and cellulitis. The onset may be insidious, however, with no evidence of lymphangitis and the condition may develop as a painless swelling. The regional lymph nodes are usually enlarged. The general appearance varies with the age of the condition. In those of long standing, the skin is thickened and leathery. The subcutaneous tissue is also greatly hypertrophied and the weight of the part often prevents all but limited motility. At times a secondary bacterial infection supervenes with the production of a septicemia.

DIAGNOSIS

The recognition and diagnosis of this condition is often easy, especially in the endemic areas. As a matter of fact, the natives in the South Pacific islands make the diagnosis of "mumu" very readily themselves. The early diagnosis is particularly of importance because this helps the prognosis considerably. One may diagnose elephantiasis due to filaria readily but it has reached a far advanced stage and nothing can usually be done. On the other hand, early filariasis, if properly handled, has a good prognosis, although no specific therapy has been found.

This condition should always be thought of in those patients with a history of having lived in endemic areas of filariasis over

any period of time. Another suggestive factor is the history of recurrent attacks of lymphangitis with fever. A history of allergic manifestations is also suggestive but lymphangitis is the first good sign of the condition.

Of course, the clinical picture of the condition as described above helps to put one on the track of the correct diagnosis but it is agreed that the most valuable single diagnostic aid is a blood film. The thick blood film should be used and the sheathed microfilariae of the periodic type can often be demonstrated in the peripheral blood at night (10 p. m. to 2 a. m.). In the non-periodic type these organisms can usually be demonstrated throughout the 24 hours. It should be recalled, however, that the microfilariae may not always be present because the parasites may not have access to the peripheral circulation, due to the anatomical location of the adult or because of the development of a pathologic sequence which prevents entrance to the blood stream. The microfilariae are readily demonstrated in the blood of the native populations of the endemic islands showing the effect of long residence in such an area. Dickson, Huntington, and Eichold[10] showed microfilariae in thick blood films of 13.6 per cent of the 1,859 natives of Tutuila examined while only 1.6 per cent of 244 children under five years of age had microfilariae.

In spite of the fact that blood studies are by far the best diagnostic aid in filariasis, they have proved of no benefit in the cases among American service men. For although careful studies have been carried out by experienced men on proved soldier cases, the microfilariae have not as yet been demonstrated in the peripheral blood. Michael and his coworkers[23] have examined several thousand thick and thin smears taken day and night but they all proved negative for microfilariae. He thinks that the microfilariae were probably present in the blood before the clinical symptoms developed.

The search for the microfilariae should not be confined to the blood stream for they are not infrequently found in the urine, ascitic fluid or in the puncture of a hydrocele.

One of the best methods of diagnosis is by means of biopsy of lymph channels and lymph nodes. This was never appreciated more than in the current cases among the service men in whom blood studies have proved nothing. These biopsy specimens are searched for adult filariae or larvae and their discovery makes the diagnosis. A reward of careful searching is seen in Michael's series in which isolated foci of living or dead parasites were demonstrated in 30 per cent of 120 biopsy specimens and of these positive biopsies, 70 per cent were in the peripheral lymph channels, the remaining in the lymph nodes. The men who get the best results with this diagnostic method suggest detailed study of the biopsies not only by hemisection but by culture, immersing in saline, and by serial sections. There are some who think that even though the organism is not found, certain histologic changes when present suggest a presumptive diagnosis of filariasis. This is still an unsettled question but further histologic studies should give the final answer.

There is also some controversy about the best site and time for biopsy. Faust[12] and some other workers do not think that biopsies ought to be taken during the acute attack while Fogel and Huntington[15] think that biopsy of the spermatic cord and regional lymphatics is absolutely unjustified. Michael and his coworkers[23] have reported, however, that removal of the upper extremity lymph nodes and lymphatics has not harmed their patients and in some instances, definitely resulted in improvement. In this matter, too, the final answer will only come with further study and experience.

A diagnostic aid of definite value and one which should not be overlooked is the roentgenogram to be used in those cases where death and calcification of the adult worms has occurred.

Another diagnostic procedure which has caused considerable controversy is the intradermal reaction as described by Taliaferro

and Hoffman, and by Fairley.[11] An antigen is used which is prepared from *Dirofilaria immitis* as described by Fairley. Until very recently, the results have not been encouraging. As a matter of fact, Hamilton in a personal communication to Napier,[26] basing his opinion on experience in the East Indies, considered the positive tests with the antigen of little value since about two out of three normal natives showed positive results. He thought that negative tests were actually of greater value in excluding filarial infection. Recently, however, Huntington,[10] using *Dirofilariae immitis* antigen from Samoan dogs, found that 83.1 per cent of 137 patients showed positive immediate and delayed reactions while Michael[23] found that 87.3 per cent of 307 proved cases showed a positive skin test. All these patients at the time of testing, had stool examinations which were negative for other nematodes. This latter fact is essential in evaluating the test because the reaction is a group reaction and is not specific for *Wuchereria bancrofti*. An even more recent report of the intracutaneous test as used by Wartman and King[33] on American troops with filariasis showed positive readings in 90.8 per cent of 164 patients. So while the exact value of the test is still debatable, we can say that numerous workers in the field believe that it is of definite value and certainly warrants further use and study.

A complement fixation test in which the antigen is also prepared from the dog filaria, *Dirofilaria immitis*, has given fairly reliable results in some hands. However, the test apparently depends on the worm being alive and it has proved impracticable under army field conditions. The technic is similar to that of the Wassermann reaction but like the skin test, it is a group reaction and is not specific for *W. bancrofti*.

The blood picture, while not diagnostic, is often suggestive and may serve as an aid. In the absence of intestinal helminth, the presence of an eosinophilia is of significance, particularly when fever is present and a chill has occurred, and the question of malaria has thus been suggested. A leukocytosis, such as often occurs in lymphangitis and other inflammatory processes which accompany filariasis and when secondary bacterial infection is present, may also aid in excluding malaria. Filariasis patients present a relative lymphocytosis but this is true of most people returning from the tropics. The red blood count in these patients is usually normal.

PREVENTION

There is considerable that can be said concerning the prevention of this disease under ideal conditions. But when we realize that these conditions do not commonly exist in the battle zones, we discover the magnitude of the problem and see why many of the attempts to control the disease have proved futile.

Since the microfilariae are mosquito-borne and the mosquito is necessary for the infection of man, we can readily see that prophylaxis should consist of: (1) the destruction of the mosquitoes and their breeding places, and (2) protection from the bites of the mosquitoes. Such a scheme of prophylaxis naturally necessitates rigid precautions and it is facilitated by the cooperation of all persons in the area. While prophylactic measures have either not been used properly or have failed in controlling the disease in some Pacific areas, there is no reason why they should not prove effective if carried out adequately and under conditions such as those seen in most regions of the United States. Therefore, it behooves us to consider the following points which have been suggested as an adequate program in the prevention of filariasis:

1. Avoid association, in so far as possible with heavily infected populations. This means in battle areas which are endemic for filariasis, establishing quarters away from the the native villages. In this way, one can avoid contact with the heavily infected mosquito population. It is probably in this respect that many of the control programs in the Pacific have been negligent.

2. Individuals harboring microfilariae in their blood should be protected by screening. This serves to protect the patients

from reinfection as well as to prevent the infection of more mosquitoes and eventually more individuals.

3. Where it is possible, efforts should be made to reduce the mosquito population by appropriate control measures. These measures should include the usual antimalarial mosquito control measures for the Anopheles and in addition, the disposal of artificial water containers, and the oiling of ditches and puddles to control the domestic Aedes and Culex.

4. In addition, screening, bed nets, mosquito repellents and sprays should be used against the adult mosquitoes. While these measures can be used on a small scale, they are somewhat impractical due to the expense involved when attempted in a large area. It is believed that the institution of a program similar to the above would prove very effective in preventing the occurrence of filariasis and in controlling its spread once cases were found.

TREATMENT

The treatment of this condition is more unsatisfactory than that of almost any other tropical disease and in spite of the tremendous strides made in the chemotherapy of other conditions, none has been demonstrated in filariasis. Since filariasis was first described, innumerable drugs have been tried, and while some have been more promising than others, all that can be definitely said at present is that treatment with drugs aimed at eradicating filarial infection is ineffective. Occasionally the treatment has resulted in a temporary decrease in the number of microfilariae circulating in the blood stream, but the adult worms were not killed and they continued to produce microfilariae.

The most promising of the chemotherapeutic substances are the trivalent antimony preparations, anthiomaline (lithium antimony thiomalate) and stibophen (fuadin). These have been tried extensively and it is thought that they may be of some antifilarial value in asymptomatic and selected early clinical cases. Brown[4] recently reported that the former drug was given intramuscularly to a series of filaria infected patients and their microfilariae count was reduced 85-100 per cent. Since this reduction was maintained for four to five months after the completion of treatment, the author presumed it to mean that a corresponding number of adult worms were killed. The actual significance of this work cannot be appreciated at the present time but we can only hope that it is either the answer to the chemotherapeutic problem of filariasis or a step in the right direction.

Several arsenicals which kill the microfilariae or prevent their migration to the peripheral circulation have been used but they are ineffective against the adult worms. Sulfonamides have also been tested and have proved to be without apparent effect. It seems that their only use lies with that of autogenous vaccines in the therapy and perhaps prophylaxis of bacterial complications.

One of the most useful clinical procedures in treatment of filarial elephantiasis of the lower extremities, is the pressure bandaging technic of Knott[21] using six-inch strips of bath toweling, painted with dextin strips and covered with cotton elastic crepe bandage and an outer muslin bandage to keep out the dirt. Exercise is required to prevent cyanosis and hasten reduction of lymphedema.

Operative procedures are thought to have a definite place late in the disease, when elephantiasis is present but they certainly leave much to be desired and constitute an admission of the failure of early treatment. The best known operative procedure is the modified Kondolean operation which surgically removes the elephantoid tissue and thus reduces the size. This reduction in size is usually temporary and recurrence is anticipated in the course of five years.

Due to the status of chemotherapy in this condition, the treatment must be largely symptomatic. For acute lymphangitis the treatment should consist of bed rest, elevation of the affected part, and scrotal support if necessary. Ice is sometimes used and local compresses of magnesium sulfate are thought to be helpful. An important

part of this initial therapy, which has been proved among the soldiers, is reassurance of the patient—reassurance that the condition is not as serious as malaria or tuberculosis, and that it cannot be transmitted to his wife and children. Such psychotherapy may prevent the psychosomatic manifestations of filariasis.

An important adjunct to the therapy is the return of the patients to some nonfilarial area because in this way, possibility of reinfection is lessened. The optimal factors of tropical humidity and temperature are removed by change to temperate climates.

PROGNOSIS

The first question everyone usually asks about filariasis is concerning the prognosis. Therefore, with thousands of men returning to the United States with this disease, it behooves us to understand fully the matter so that we can prognosticate wisely.

In the first place, the prognosis for life is generally fair to good. Even if the patient should develop elephantiasis, apart from the disability produced, the outlook is good because the condition is a chronic one and patients frequently live for years. Such a prognosis, of course, does not hold in cases with secondary bacterial invaders which produce a septicemia.

The acute stage of the disease subsides after a few days' rest but the patients should not return to full duty for 10 to 14 days after an acute attack or even longer if the scrotum is involved. Even then, the resumption of activity should be very slow because recurrences with renewed activity are not uncommon. It is a well recognized fact that recurrences are much more common in Samoa and the other endemic areas than they are when the patient is moved back to the United States. This has led us to believe that the prognosis is much better if the patient is removed from the endemic area after an acute attack.

It is felt by Dickson, Huntington, and Eichold[10] that there is little risk of deformity of the legs and arms with an acute attack of filariasis but deformity of the scrotum has been recorded and the prognosis in such cases is doubtful. Michael, however, after careful studies of the pathology of the acute cases among American troops, stated that the pathologic lesions appeared to be reversible and that after the filarial reaction has subsided, even the genital lesions should return to normal.

One of the important questions considered in giving a prognosis is the matter of sterility. Since the pathology is often genital, it is a natural question to be raised and this problem often constitutes the major concern of the young men involved. Unfortunately one cannot say at the present time with certainty whether this condition will produce sterility to any appreciable degree, because no data are available as to the incidence of sterility in filarial epididymitis and vasitis. We can say, however, that the native populations in the endemic areas appear relatively prolific and this fact speaks against sterility due to filariasis. Michael thinks that if sterility develops, it should be of a temporary nature and that the genital lesions should return to normal.

It seems that a better insight into the matter can be gained by considering Napier's[25] ideas concerning the disease. He believes that the psychologic trauma that our troops have endured far exceeds in seriousness, the somatic trauma; that they have seen the bizarre deformities suffered by a large population of the natives in the hyperendemic islands in which they acquired their own filarial infections and that they have not unnaturally assumed that their fate would be the same. He further points out that due to the weakness of medical texts to emphasize the extremes of disease, the ordinary physician is likely to believe that filarial infection is inevitably associated sooner or later with a huge scrotum or an elephantoid leg, while the contrary is the truth. In certain endemic areas, for instance, even though 5 per cent of the native population may exhibit filarial infection, only a fraction of 1 per cent shows gross filarial lesions at any time during their lives. It is important to remember that obstruction of the lymphatics of a

whole limb occurs only after repeated heavy infections over a long period of time.

The question that arises then, which we must answer, is whether the doctor can honestly assure returning service men, who have been subjected to heavy filarial infection, but for limited periods of time, that they will not suffer from any serious elephantoid deformities and that their sexual powers will be unimpaired. After considering the facts which have been presented, I agree with Napier that a favorable prognosis can be given this condition with as much assurance as any medical prognosis can be given.

FUTURE HEALTH PROBLEM IN UNITED STATES

After reviewing filariasis briefly in the light of some of the newer concepts which have been emphasized since our entry into the war, it seems fitting to attempt to determine the status of the condition in the United States, not only for today but for tomorrow as well. We should attempt to answer the question, "Will filariasis constitute a public health problem in the United States in the future?"

There is no one who can deny that the filariasis problem exists as a possibility in the future. Theoretically we have everything in the United States which is necessary for the condition to become endemic. The only factors needed for transmission of the disease are the proper vectors, a source of microfilariae, and a susceptible population. We have many species of mosquitoes in the United States belonging to the genera Culex, Aedes or Anopheles which are the intermediate common hosts and vectors of the filarial organisms. We also have a sizable potential source of microfilariae in the service men returning from the battle areas and the susceptible population is certainly provided by the American public. But in addition to these factors which fulfill the requirements for transmission of filariasis, there is one other item which points even more conclusively to the condition as a post-war problem. This is the fact that there have been proved endemic foci of the disease within the continental United States. Such a consideration somewhat removes the filariasis problem from the realm of pure theory because these foci show beyond any doubt what can and may happen in the United States.

But in spite of the fact that all of these things speak strongly for the possibility of filariasis as a future problem, there seem to be even stronger arguments against such a probability. In the first place, it is important to note that in those hyperendemic areas where the disease constitutes a major health problem, the chronic cases which are represented by elephantiasis in one form or another, only result after repeated infections by a heavily infected mosquito population. In other words, the acute cases which are the variety which have been brought to the United States, are not thought to become chronic without additional reinfection. In fact, some workers believe that many of the victims of acute attacks will never have a recurrence. This conclusion has been reached only after a careful study of the pathology of the disease. Michael, for example, believes that the natural defenses of the body, the removal of the patient from endemic areas, and the prevention of reinfection will result probably in the disease running a self limited course.

One of the most significant arguments against filariasis as a major health problem in this country is the fact that numerous trained workers have searched diligently for the microfilariae in the peripheral blood of proved filarial patients without results. This is an excellent indication that the potential source of microfilariae in this condition may never become an actual one. Of course, there is nothing definite about this because most writers agree that in these patients, the microfalariae have probably been present at one time or another. The fact, however, that they have not been demonstrated as yet is certainly hopeful.

It seems that a good approach to this situation is to study one of the previous endemic foci in the United States. In Charleston, South Carolina, for instance, a definite focus of endemic filariasis was re-

ported by Francis[16] in 1919. Of even greater significance to the point under consideration is the fact that this focus no longer exists. For whatever the cause of the disappearance of this focus, it offers a tentative solution to a future problem should it become acute. Unfortunately, there does not seem to be a great deal in the literature concerning the measures undertaken to control this focus. It is presumed that the control was brought about through improved and rigid mosquito control measures, and this certainly gives us an idea around which to build our preventive measures. Undoubtedly, however, the fact that the optimal factors for the disease (tropical humidity and temperature) are not present here in the temperature climates, played an important part. So, even if foci of filariasis should develop, they are likely to die out, just as happened in Charleston where the focus existed for a century and a half and yet did not spread to other parts of the country.

In the light of these arguments, it seems that we can relegate filariasis as a post-war problem, to a place of little importance. But no matter how convincing evidence seems to be against the probability, we must always keep in mind that establishment of disease foci here is a definite possibility. At the same time, we are comforted by the fact that mosquito control in this country is far better than that of any endemic area of the disease and adequate mosquito control will not only aid in controlling foci which exist but will also prevent their establishment.

One other matter to be considered in discussing filariasis as a post-war problem, is the possibility of individual service men, who have contracted the disease, becoming an economic problem to the government. It has been noticed by some that the men after an acute attack may have a recurrence of the symptoms when they resume their normal activity. How long these recurrences will continue to take place and whether they will prevent these men from returning to normal work after the war is not definitely known at present. It has been called to our attention that the recurrences seem to get further apart and the periods of relapse shorter in duration but the final answer cannot be given at this time. If these men were incapacitated due to recurrence, they would be candidates for pensions from the government and this might be no small problem at the present rate of increase of filariasis cases.

SUMMARY

This paper gives a brief discussion of the geographic distribution, etiology, mode of transmission, pathology and pathogenesis, symptomatology, methods of diagnosis, prevention, treatment and prognosis of filariasis, in the light of some of the newer concepts of the disease which have been formulated since the war began.

With these concepts and ideas of the disease before us, we have discussed the possibility of filariasis becoming a future problem in the United States. It is believed that the possibility of filariasis existing as a post-war problem is definite because the United States has all the factors necessary for the transmission of the disease: suitable vectors, a source of microfilariae, and a susceptible population. It is also pointed out that certain endemic foci of this disease have already existed in this country.

In spite of these arguments, it is felt that filariasis will not become an important or serious post-war problem because: (1) The source of microfilariae is only potential, not actual at the present time; (2) there is little danger of acute cases proceeding to chronic ones here because repeated reinfections are necessary by a heavily infected mosquito population; (3) acute cases when returned to this country have fewer recurrences and fare better than those remaining in the hyperendemic areas; and (4) a study of previous endemic foci in this country show that they died out and did not spread with the institution of adequate mosquito control.

Therefore, it is felt that filariasis can be relegated to a place of little importance as a future problem, although the potentiality of the disease should not be overlooked.

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NEW ORLEANS

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DEFERMENT OF THE ANNUAL STATE MEETING

We regret very much that the Office of Defense Transportation has forbidden the annual state meeting which was to be held on April 13. Likewise they have also forbidden the holding of the New Orleans Graduate Medical Assembly in the same week. Of course if the O.D.T. thinks that the holding of these two meetings will impinge in any way upon our efforts to win the war, we gladly acquiesce in their ruling. It does seem, however, that meetings which are purely scientific and instructive to the physician and which in a sense are very largely local, should not come under the ban of the Office of Defense Transportation.

The state meeting has not been called off but has been indefinitely postponed. When there occurs relaxation in the travel regulations, then there will be a meeting called by the Executive Committee. In the meantime it is to be hoped that the district and parish medical societies will carry on vigorously and actively not only in science but also in free discussion of the economic and social problems that face the medical profession. Never in the history of medicine of the United States was there a time when doctors should stick together as closely as at the present time. We must be prepared to act as a unit in opposing many of the projects from the Congress which will result in loss of our individuality as a profession and our regimentation as doctors.

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ANNUAL MORTALITY SUMMARY

It is a remarkable thing that in wartime when epidemics are more likely to be rife, when there exists a shortage of physicians, that the number of deaths in 93 major cities of the United States decreased almost 4 per cent as compared to the previous year. Of the 93 cities that reported to the Department of Commerce, Bureau of Census, in the year 1944 there were 468,773 deaths as compared with 487,931 in 1943. This decrease in the total number of deaths was furthermore notable because in January of 1944 there occurred an epidemic of influenza and pneumonia which very materially increased the number of people dying in that month. There were over 4000 more people who expired this month than would be anticipated from the three-year average.

It is impossible to compare different cities because the death rates have not been computed. As the figures that are submitted weekly by the city health departments do not take into account the population growth or decline, it is also impossible to evaluate the number of deaths on

an actual population basis. In many cities populations have increased materially by an inflow of war workers from the rural districts of the country. In some cities war work has not been carried on to any very great extent and in some cities the population has actually diminished.

Although it is not possible to give percentage differences for the cities in the South, it might be of some interest to note that in nearly all of the Southern cities the number of people dying has been materially reduced. In the City of New Orleans there was a total of 793 deaths in 1944, some 213 less deaths than in 1943. Atlanta, with a smaller population than New Orleans, showed a diminution in the number of deaths amounting to 247. Birmingham had 121 less deaths, Louisville 230, Memphis 153, Nashville 16 and Houston 102. There were a few Southern cities which showed an increased number of deaths among their citizens. This was notable in the two cities that report from Florida—Tampa and Miami. In Richmond the number of deaths increased as it did also in Chattanooga and Knoxville.

The death rate in New Orleans will always be higher as contrasted with other large Southern cities because mortality figures are tabulated on the basis of the place of death and not of residence. Many of the deaths in the City of New Orleans, as is well known, are of people who have been imported into the city to be patients in the Charity Hospital. The death rate in New Orleans will undoubtedly be the lowest it has been for some years for two principal reasons: (1) because there has been a remarkable population increase on account of war industries located in the city and (2) because many fewer people have been admitted to the Charity Hospital than when the hospital was at its peak in population. The decrease in the number of deaths in the city reflects credit on the state and on the municipal health departments of the state. Also it probably reflects the result of the newer chemotherapeutic agents which have lowered so materially the number of deaths in people with certain of the infectious diseases.

Particularly commendable is the lowering of infantile mortality. There were over 2000 fewer children dying in the 93 cities that have been tabulated, than in 1943. A complimentary statement cannot be made in regard to the infant deaths in New Orleans, as there was an increase of 18 deaths in white infants in the city, or a 2.7 per cent increase, and 28 more deaths in colored babies, in terms of per cent, 7.7. One wonders if this infantile mortality increase in New Orleans may not be due to many young mothers working in war industries or in civil occupations who, prior to the demand for labor, would have been at home to attend to their small children.

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GRADUATE INSTRUCTION FOR RETURNING MEDICAL OFFICERS

The young physician who has completed nine months of internship and then entered the armed forces feels very definitely that his training has not been sufficiently adequate to fulfill his needs. The same statement applies to some of the men who had nine months or even 18 months of training in one or another of the specialties but who have not had time enough to qualify themselves for a specialty board examination. The feeling that many of the young medical men in the armed services have now is that they will not have had training to qualify them in a specialty nor will the required facilities be available to make it possible for them to have such training if they want it.

There are also a large number of men who have been in practice a comparatively short time who have not had the opportunity of working in Army or Navy hospitals and who feel that they will need further training. In this group, as well as in the group of recent graduates, the fear that they will have forgotten much of their medical information is certainly distinctly lowering to the morale of these medical officers on duty in the field. However, it may be said with the utmost confidence that the

plans which have already been worked out will provide for nearly every medical officer who wishes to have either a short or a long period of further training. To those who wish three to six months' refresher courses, such courses will be given in medical schools and in the larger hospitals. To the men who wish to return for more prolonged training, notably as residents, the hospitals throughout the country are preparing to offer very many residencies in addition to those already established.

The responses to the questionnaire sent out to the medical officers in the Army and Navy through the American Medical Association, indicate that in many of the specialties, residencies in subjects such as anesthesia, dermatology, neurosurgery, neurology, psychiatry, pathology and radiology may be taken care of without extension of the already existing facilities. On the other hand, in internal medicine, obstetrics and gynecology, otolaryngology and most notably surgery, extra residencies will have to be set up. So far as now ascertainable, residencies will and can be provided in number ample to take care of the young medical officer who wishes to go into any of these fields except that of general surgery. It would seem that a very large proportion of young men want to specialize in surgery. From the present indications there will not be enough additional surgical residencies available to begin to take care of all who wish this training. It is to be presumed that it may be possible for the surgical board to reduce the number of years required for surgical training in order to give opportunity for training to these surgeons in embryo, particularly in view of the fact that many of them have had extensive experience in traumatic surgery. There is one disturbing feature to which much thought should be given and that is that about eight out of every ten young men in the service want to become a specialist when they finish their tour of Army duty. From whence will come the general practitioners of the future?

THE TREATMENT OF VINCENT'S ANGINA

Vincent's organisms are commonly found in the mouth of about 75 per cent of adults. In the majority of these people there are no symptoms. In a small group there will be evidence of a stomatitis and in an even smaller group there will be an ulcero-necrotic lesion or lesions on the tonsils or on the buccal mucous membrane. There may be merely local expressions in these people but from time to time there is a systemic reaction associated with fever and the symptoms that are associated with a rise in temperature. In a very few instances the reactions are very severe. This has been called colloquially systemic or constitutional Vincent's and from this primary mouth condition patients may die. It should be noted also that not only in any dirty mouth but also in nearly every subacute or chronic oral condition the Vincent's organisms will be found, although they may not be responsible for the mouth lesions. Oral cancer is an excellent example as is agranulocytic angina.

The treatment of Vincent's angina has been singularly unsuccessful. Illustrative of the poor results obtained in the treatment of Vincent's is the multiplicity of therapeutic measures that are advocated by various physicians. In the milder cases with a marked stomatitis, daily visits to a dentist with local applications of arsphenamine on cotton, between and around the teeth may be effective, but this is a time-consuming and prolonged form of treatment.

On account of the difficulty of treating Vincent's angina satisfactorily and the unsatisfactory results that are usually obtained, a very short but well worth-while report by Lt. Commanders Manson and Craig* may have been missed by readers of the Journal of the American Medical Association. These two Naval officers report on 48 patients with severe angina who

*Manson, W. W., and Craig, I. T.: Treatment of Vincent's angina with sulfathiazole, J. A. M. A., 127:277, 1945.

were treated with sulfathiazole tablets. The condition responded promptly to treatment and recurrences did not occur. The one-half gram sulfathiazole tablets are allowed to dissolve in the mouth every two hours during the day and every four hours at night. This treatment was continued for 72 hours except in an occasional mild case when it was continued for only two days. The author says that the lesions were invariably cleared up within a period of 96 hours after the beginning of treatment. This short report should focus the sulfa treatment of Vincent's angina on the mind of the medical profession. Treatment is relatively simple and the results are, according to the two Naval officers, 100 per cent perfect. Undoubtedly many members of the medical profession have made use of sulfa tablets in mouth conditions but this report of Manson and Craig is the first to put on record the successful treatment of a large number of patients with an often intractable and difficult lesion to treat.

ORGANIZATION SECTION

The Executive Committee dedicates this page to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

THE MEETING OF THE EXECUTIVE COMMITTEE

The Executive Committee of the Louisiana State Medical Society held a meeting on February 10. It may be of interest to members of the profession to know some of the important transactions that occurred at this meeting.

The status of the poliomyelitis clinic, which is being conducted by the Louisiana State Board of Health at the Charity Hospital in New Orleans, was brought to the attention of the Executive Committee by Dr. David Brown, Director of the State Board of Health. After considerable discussion the following motion prevailed: "That the Executive Committee of the State Medical Society agrees to the continuance of the poliomyelitis clinic, recommending that it be placed under the jurisdiction of Charity Hospital, providing proper financing can be done without federal aid."

Considerable attention was given to the material which we have been receiving in our office of the State Medical Society from Congressional sources and from the American Medical Association's Committee of Medical Service and Public Relations, especially in relation to the various attempts made by Congress to control various phases of the practice of medicine. It was felt by the State Council that a Congressional Committee should be appointed to review this material and make appropriate recommendations to the executive officers of the State Society. It was thought in this manner adequate opposition could be brought to bear on bills emanating in Congress antagonistic to our idea of the American way of practicing medicine, and that our Society would be able to assume a more positive action in relation thereto. Postwar planning was discussed very liberally, and a committee will be appointed to digest and implement the facts as revealed: First, from a questionnaire sent to the doctors in the military service upon their needs on returning to civilian practice; second, the Bureau of Information of the American Medical Association in cooperation with the Office of the Surgeon General of the Army is sending through our office to every parish a comprehensive questionnaire for factual data on prevailing conditions as to medical facilities, hospitals, number of doctors; third, the State Society will send out to each doctor of the state a questionnaire to obtain some information as to the needs of the physicians now in civilian practice in relation to medical facilities,

need for additional medical assistance, and other recommendations which would be helpful to the doctors returning from military service. You can see from the above that the Executive Committee is attempting a serious and conscientious effort to obtain suitable and accurate data for the aid of the returning physicians from a professional, educational, and economic viewpoint. These efforts will be entirely fruitless unless we have the assistance of every doctor in Louisiana. It is felt that all now engaged in civilian practice would be only too glad to aid our fighting confrères in getting reestablished in civilian practice.

The following letters in regard to the postponement of the date of the annual meeting of the House of Delegates on April 13 are self-explanatory:

Office of Defense Transportation
Washington, D. C.

February 3, 1945

Louisiana State Medical Society
Dr. P. T. Talbot, Secretary-Treasurer
1430 Tulane Avenue
New Orleans 13, Louisiana

Dear Dr. Talbot:

Your application for permit to hold a conference in New Orleans at the Hotel Roosevelt on April 13 has been reviewed by the Committee.

It is the consensus of the Committee that this meeting should be deferred until such time as the necessity for the present restrictions ceases to exist. Permit is therefore denied.

Very truly yours,
(Signed) R. H. Clare, Secretary
War Committee on Conventions

St. Clair Adams and Son
Attorneys and Counselors at Law
New Orleans

February 6, 1945

Dr. Val H. Fuchs, President
Louisiana State Medical Society
New Orleans, La.

Dear Doctor:

Referring to your request for an opinion with reference to the functioning of the Association because the United States authorities in Washington have held that your Association cannot hold its annual meeting, as provided by Article V of your Charter, I beg to advise that, in my opinion, the Association should continue to perform its objects and purposes with the officers elected at the last annual meeting until such time as the Association can hold an annual meeting, in accordance with the terms of its Charter.

We are in a state of war, as you know, and the Government has determined that it is for the general welfare not to permit the holding of annual meetings and conventions or to otherwise burden transportation facilities and while it works a hardship upon many associations similar to yours, nevertheless it does not in any manner affect the integrity of your Association, because the subject-matter is completely governed by the article of your Charter just referred to, where it is said:

"The officers elected at the last annual meeting shall hold office under this Charter *until their successors are elected and installed;****

Under your Charter the officers can only be elected at an annual meeting and naturally if such a meeting cannot be held they cannot be elected and consequently this language comes into play and the current officers must continue to act until their successors are legally elected and installed, in accordance with the dictates of an annual meeting.

With expressions of high esteem and personal regards, I beg to remain,

Yours sincerely,
(Signed) St. Clair Adams

The Executive Committee adopted unanimously this opinion of the Honorable St. Clair Adams in regard to postponement of the meeting. Just as soon as the necessity for this ruling of the Office of Defense Transportation is removed, the Executive Committee will arrange for an early date for a meeting of the House of Delegates, in order that they may transact the necessary business of our organization.

Owing to the fact that the date of the meeting of the House of Delegates is an uncertain one, the Executive Committee has requested that each member of the profession be sent a copy of the financial statement for 1944 and a copy of the budget for 1945. Just as soon as this material can be correlated in an appropriate form it will be sent, and it is hoped that you will take the occasion to review same.

We are thus confronted with the great problem of carrying on our organization under the trying circumstances of a delay in the usual performance and transactions of business by the House of Delegates. It is therefore imperative that the individual doctors in our parish and district societies try in every manner to help to maintain and also to perfect suitable plans for the great work ahead of us. This is a time when our parish and district societies should, if possible, meet regularly and to have on their programs topics concerning medical service of the future or other vital medical subjects for the welfare of our organization and the civilian population.

Without any attempt to direct or impose any particular subject, we would like to take the opportunity of directing your attention to a few things which could be discussed profitably by the parish and district societies. One of the most important is the postwar plan which was spoken of in the above resume. Another is the question of establishing in our state some form of voluntary prepayment medical insurance. This subject is receiving the attention of a very active committee, and it is known that they were prepared to make a very instructive report to the House of Delegates on this vital subject.

There are many matters of national interest emanating from Congress. In the Organization Section of February, we brought to your attention the introduction in Congress of H. R. 395 bill by Representative Dingell. It should be of interest to you to know that recently there was introduced by Representative A. L. Miller of Nebraska H. R. 1391, a bill to create a Department of Health in the Cabinet. It is believed that the medical profession is vitally interested in this bill, as it attempts to establish in the Cabinet a health officer, which for many years has been the object of the medical profession of this country. An antivivisection bill, H. R. 491, should receive our opposition as it attempts to make it impossible to carry on animal experiments in the District of Columbia. In the February issue of the Journal we mentioned the Pepper report which represented the views of a Sub-committee of Postwar Education and Health of the Senate. It might be of interest to know that springing from this report is the Senate Bill 191 known as the Hill-Burton bill implementing the Pepper report. This legislation, if passed, attempts to make a survey of medical and clinical facilities over the United States, construction of hospitals and needed beds, all at a state level. These measures spoken of above are very vital to the medical profession, and every doctor should contact his representatives and senators expressing his views concerning same.

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TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

March 1. Clinico-pathologic conference, Touro Infirmary, 12:00 noon.

March 5. Board of Directors, Orleans Parish Medical Society, 8 p. m.

March 6. Eye, Ear, Nose and Throat Staff, 8 p. m.

March 7. Mercy Hospital Staff, 8 p. m.

March 12. Scientific Meeting, Orleans Parish Medical Society, 8 p. m.

March 14. Woman's Auxiliary, Orleans Parish Medical Society, Orleans Club, 3 p. m.

Clinico-pathologic conference, Marine Hospital, 7:30 p. m.

Touro Infirmary Staff, 8 p. m.

March 15. Clinico-pathologic conference, Touro Infirmary, 12:00 noon.

March 16. I. C. R. R. Hospital Staff, 12:30 p. m.

March 19. Hotel Dieu Staff, 8 p. m.

March 20. Charity Hospital Medical Staff, 8 p. m.
March 21. Charity Hospital Surgical Staff, 8 p. m.
March 22. DePaul Sanitarium Staff, 8 p . m.
March 23. L. S. U. Faculty Club, 8 p. m.
March 27. Baptist Hospital Staff, 8 p. m.
March 28. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
French Hospital Staff, 8 p. m.
March 29. Clinico-pathologic Conference, Touro Infirmary, 12:00 noon.
March 30. New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

During the month of February the Society held one regular scientific meeting. The following program was presented: Malignant Diphtheria—A Case Report by Dr. Charles L. Cox; A Case of Rectovaginal Fistula by Dr. Robert F. Sharp; Temporal Approach in Depressed Fracture of Malar-Zygomatic Compound by Dr. Waldemar R. Metz; Fever of Psychic Origin by Dr. Oscar W. Bethea; A Case of Perirenal Abscess by Dr. E. A. Ficklen; An Unusual Case of Ureteral Calculi—Five Stones in One Ureter by Drs. Hugh T. and W. D. Beacham.

NEWS ITEMS

Dr. Theo J. Dimitry was one of ten Loyola faculty members to receive a citation for services to that university for over twenty-five years.

Dr. Frank E. Lamothe was recently elected assistant rabban of Jerusalem Temple of the Shrine for 1945, and Dr. Abe Mattes was elected high priest and prophet.

Dr. Henry Ogden recently attended a meeting of the American Academy of Allergists in New York.

Dr. Daniel N. Silverman spoke on the importance of the role of protein and vitamin therapy in gastro-enterologic disorders at the monthly meeting of the New Orleans Dietetic Association recently held at Charity hospital.

Dr. H. Ashton Thomas and Dr. Louis A. Wilensky recently received certifications by the Board of Otolaryngology.

Dr. Wilbur C. Smith was elected president of the National Collegiate Athletic Association at the closing session of the association's annual conference at Columbus, January 13. In the fifty-year history of the association Dr. Smith is the first representative of a Southern university to occupy the top executive position.

The Southwest Allergy Forum will hold its annual meeting at the Jung Hotel on April 5-10.

Dr. Hilliard E. Miller attended the meeting of the American Gynecological Club in Baltimore, February 23-25. Dr. Miller is president of this organization.

At a recent meeting of the Orleans Parish School Board Dr. Emile A. Bertucci, Sr. was promoted from examining physician to director of field activities in the Department of Hygiene and Child Welfare, New Orleans Public Schools.

At the January quarterly meeting of the Catholic Physicians' Guild the following officers for 1945 were elected:

Dr. Edwin L. Zander, president; Dr. N. F. Thiberge, vice-president; Dr. Theo F. Kirn, secretary; Dr. P. A. Boudreaux, treasurer; Drs. Joseph A. Danna, E. L. Leckert and E. J. Richard to the executive committee; Dr. Henry Ogden, chairman of membership committee; Dr. Ruth Aleman, chairman of activities.

Dr. Frank Chetta was installed as president of the medical staff of Hotel Dieu at the annual dinner meeting held January 15. Other officers installed were: Dr. Lucien A. Fortier, vice-president; and Dr. Hugh T. Beacham, secretary-treasurer; Dr. W. A. Gillaspie, Dr. Monte F. Meyer and Dr. C. Walter Mattingly, members of the board. Dr. Chaille Jamison, the retiring president, was toastmaster for the dinner.

Dr. N. J. Tessitore was installed as president of the staff of Mercy hospital at the annual dinner meeting held January 3. Other officers installed were: Dr. William H. Roeling, vice-chairman; Dr. C. J. Vedrenne, secretary; and Dr. Everett L. Drewes, treasurer; Dr. R. J. Mailhes and Dr. Louis Monte, members of the board.

Dr. Edmund Connely was recently appointed as city alienist and ex-officio head of the city hospital for mental diseases.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria

Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

ASCENSION PARISH MEDICAL SOCIETY

At a recent meeting of the Ascension Parish Medical Society the following officers were elected for the year 1945: President, Dr. E. S. Kyes, Dutch Town; Vice-President, Dr. Dawson T. Martin, Donaldsonville; Secretary-Treasurer, Dr. E. A. Schexnayder, Donaldsonville; Delegate, Dr. Percy LeBlanc, Donaldsonville; Alternate, Dr. Myer Epstein, Gonzales.

ASSUMPTION PARISH MEDICAL SOCIETY

The following officers were elected for the year 1945 by the Assumption Parish Medical Society: President, Dr. Henry A. LeBlanc, Paincourtville; Vice-President, Dr. H. C. Dansereau, Labadieville; Secretary-Treasurer, Dr. Julius W. Daigle, Paincourtville; Delegate, Dr. Julius W. Daigle; Alternate, Dr. C. S. Roger, Napoleonville.

CALCASIEU PARISH MEDICAL SOCIETY

The Calcasieu Parish Medical Society met on Tuesday, January 9, 1945, at 7:30 p. m. at the St. Patrick's Hospital, and the following officers and delegates were elected unanimously for 1945: President, Dr. W. A. K. Seale, Sulphur; Vice-President, Dr. W. P. Bordelon, Lake Charles; Secretary-Treasurer, Dr. Eleanor Cook, Lake Charles; Delegate, Dr. G. C. McKinney, Lake Charles; Dr. W. A. K. Seale, Sulphur; Alternates, Dr. W. G. Fisher and Dr. Walter Moss, both of Lake Charles.

LAFAYETTE PARISH MEDICAL SOCIETY

Newly elected officers of the Lafayette Parish Medical Society for 1945 are as follows: President, Dr. L. A. Prejean, Scott; Vice-President, Dr. C. J. Saloom, Lafayette; Secretary-Treasurer, Dr. A. A. deMier, Lafayette; Delegates, Dr. E. E. Guilbeau, Carencro and Dr. Paul Kurzweg, Lafayette; Alternates, Dr. J. M. Miles, Lafayette and Dr. L. O. Clark, Lafayette.

SHREVEPORT MEDICAL SOCIETY

The following are the newly elected officers of the Shreveport Medical Society for 1945: President, Dr. W. R. Mathews; First Vice-President, Dr. J. E. Heard; Second Vice-President, Dr. C. H. Webb; Secretary, Dr. T. B. Tooke, Jr.; Treasurer, Dr. E. B. Flake; Historian, Dr. J. M. Bodenheimer; all of Shreveport.

ST. MARTIN PARISH MEDICAL SOCIETY

At a meeting of the St. Martin Parish Medical Society held on January 19, Dr. J. L. Beyt of St. Martinville was elected president, and Dr. S. D. Yongue of Breaux Bridge was elected secretary-treasurer for 1945.

LAFOURCHE PARISH MEDICAL SOCIETY

On January 24, 1945, at Delaune's Restaurant, Lockport, La., a regular meeting of the Lafourche Parish Medical Society was held.

The program was begun by the election to membership in the society of Dr. Tom Kleinpeter of Thibodaux, La. and Dr. Philip Robichaux of Raceland, La.

This was followed by election of officers for the year 1945. The following members were elected: President, Dr. J. L. Danos; Vice-President, Dr. L. Chatelain; Secretary-Treasurer, Dr. P. A. Robichaux.

Dr. T. Benton Ayo was elected as delegate to the state convention with Dr. L. J. Kerne as alternate.

A discussion in regard to the merits of the association of American Physicians and Surgeons Inc. followed. It was decided to await until after the State society meeting before formulating a parish policy in regard to the above.

A motion by Dr. J. L. Danos to write the A. A. P. S. of the intentions was passed.

By unanimous vote a motion was passed to contact the state society requesting inactive membership for Dr. P. J. Dansereau who had been Secretary-Treasurer of the society since 1918.

Roll Call: Dr. T. B. Ayo, Dr. Charles J. Barker, Dr. C. Chatelain, Dr. J. L. Danos, Dr. Guy Jones, Dr. L. J. Kerne, Dr. P. A. Robichaux.

Dues for local chapter of $1.00 were paid by the above.

There being no further business the meeting was adjourned.

Philip Robichaux,
Secretary-Treasurer.

FIFTH ARMY NEWS RELEASE

TULANE-SPONSORED HOSPITAL IN ITALY AWARDED FIFTH ARMY PLAQUE

With the Fifth Army, Italy—The 24th General Hospital, created in New Orleans, Louisiana, by Tulane University School of Medicine, recently was awarded the Fifth Army Plaque and Clasp for exceptionally meritorious performance of duty in the Italian war theater.

The citation accompanying the award, signed by Lieutenant General Mark W. Clark, 5th Army Group commander, stated:

"This organization provided superior medical attention to thousands of Fifth Army troops. The 24th General Hospital maintained highest stand-

ards of professional treatment despite heavy emergency demands which were made repeatedly. The noteworthy accomplishments of this hospital reflect the high traditions of the Medical Corps of the United States Army."

The hospital unit is commanded by Colonel Walter C. Royals, 60 Farnham Place, New Orleans, a graduate of the Tulane medical school. Most officers of the hospital are former faculty members or graduates of the Tulane school, and the majority of its nurses formerly practiced at either Charity Hospital, New Orleans, or Touro Infirmary, in the same city.

SYMPOSIUM OF TUBERCULOSIS

There was to have been held in New Orleans on April 17-19, a symposium on tuberculosis which was to have been participated in by numerous well-known students of this disease. Dr. Chester A. Stewart was in charge of the program, and he had secured a group of men well-known throughout the country for their studies on tuberculosis. The symposium was sponsored jointly by the Louisiana State University School of Medicine, the National Tuberculosis Association, the Tuberculosis Control Division of the United States Health Service, and the Louisiana State Tuberculosis Association. The meeting promised to be of great importance, but unfortunately, in spite of the fact that the number of guests was to be limited to thirty-nine, the ODT ruled that the meeting could not be held.

TOURO INFIRMARY

The regular meeting of the Medical Staff of Touro Infirmary was held on Wednesday, February 14 at 8 p. m. The first presentation on the program was a clinico-pathologic conference led by Dr. Thomas Findley.

This was followed by a symposium on penicillin. Dr. Rawley M. Penick first presented a report of the penicillin committee and then the superintendent, Dr. Lewis E. Jarrett, discussed penicillin rationing. Following these discussions a symposium on surgical infections, medical conditions, penicillin in gynecology and obstetrics, in otolaryngology, in genito-urinary condition and in pediatrics was presented by Drs. Rawley M. Penick, Manuel Gardberg, Hilliard E. Miller, Harold Kearney, Melvin Gold and J. D. Russ, after which there was a general discussion of the use of penicillin.

The program was completed by a talk from Mr. J. B. Rivers of Mead's on the composition and use of amigen in medical and surgical procedures.

SOUTHERN BAPTIST HOSPITAL

The Southern Baptist Hospital staff presented a program which was given before the Orleans Parish Medical Society on Monday, February 12. Those appearing on the program included Drs. Charles L. Cox, Robert F. Sharp, Waldemar R. Metz, Oscar W. Bethea, E. A. Ficklen, Hugh T. Beacham, and W. D. Beacham.

The regular monthly clinical staff meeting was held on January 23 at 8 p. m. The program consisted of a paper entitled Observations on Uses of Thiouracil by Dr. P. M. Tiller. Dr. W. H. Gillentine gave a case report on Renal Complications of Sulfadiazine. Succeeding these presentations came the monthly death report by Dr. J. W. Wells, which was followed by election of officers, the following members of the staff being elected: Dr. Sam Hobson, chairman; Dr. John T. Sanders, vice-chairman; Dr. J. W. Wells, treasurer, and Dr. W. H. Gillentine, secretary.

NEWS ITEMS

Dr. Harry A. Davis, Associate Professor of Surgery, Dr. John S. LaDue, Assistant Professor of Medicine, Louisiana State University School of Medicine on the Louisiana State University Unit at Charity Hospital attended the regional meeting of the American Federation for Clinical Research held in Dallas, Texas, February 2 and 3, 1945. Dr. Davis presented a paper entitled "A comparative study of thrombo-angitis obliterans in white and negro patients"; Dr. LaDue talked on "Possible explanation of the mechanism of compensation of the failing human heart"; and Dr. Levy gave a paper concerning "Results in the treatment of subacute bacterial endocarditis employing combined penicillin and heparin therapy".

Dr. Edgar Hull, Professor and Director, Department of Medicine, and Dr. Louis Monte, Clinical Associate Professor of Medicine, Louisiana State University School of Medicine attended the Regional Meeting of the American College of Physicians at Memphis, Tennessee.

Dr. Hull who is College Governor for Louisiana presented a paper entitled "Bacterial Endocarditis" and presided at the afternoon session.

The Director of the Pan American Sanitary Bureau advises, at the request of the Cuban Embassy in Washington, that the Second National Cancer Congress will be held in Havana May 5-21, and the Organizing Committee extends a most cordial invitation to any American physicians who may wish to attend and especially to those interested in this disease.

UNDULANT FEVER

The Journal is in receipt of a letter from Charles M. Jacob, D. V. M., Convent, Louisiana, in reference to the article which was published in the Journal by Dr. Harry J. Schmidt. Dr. Jacob points out that his own son has undulant fever; that it is an extremely severe disease; and that there should be extensive research in the pro-

gram of brucellosis so that a cure could be found for the disease.

Dr. Jacob also points out that Bang's disease is very prevalent in the parish and that the people should not be allowed to drink raw milk. He says that every milch cow should be tested for tuberculosis and Bang's disease "as soon as a calf is born."

NEED FOR PHYSICIANS IN THE NAVY

The serious need for physicians in the U. S. Naval Reserve is emphasized in recent communications from the Bureau of Naval Personnel of the Navy Department.

Since the Army discontinued the commissioning of physicians, it was anticipated that the procurement of physicians by the Navy would be increased. Actually the number of physicians commissioned in the U. S. Naval Reserve has been decreasing. Three thousand physicians are needed as soon as possible to ease the emergency which now exists. Even this number will not actually satisfy the demand.

Your state has already contributed more than its share of physicians to the various services, but it is necessary to secure every physician who is not absolutely essential to the health and welfare of your state.

Many physical defects may be waived for commission in the U. S. Navy Medical Corps Reserve. This is being done in order to help fill the urgent need for medical officers. The age limit is now 55. Doctors up to the age of 60 may apply for commission and be assigned to the U. S. Veterans Administration. Rank is based on both age and experience.

Any applicant should consult the State Chairman of Procurement and Assignment Service for Physicians regarding his possible release and then contact the Director of Naval Officer Procurement, 611 Gravier St., New Orleans, La.

FROM THE LOUISIANA NURSING COUNCIL FOR WAR SERVICE

TO THE LOUISIANA STATE MEDICAL ASSOCIATION

Louisiana physicians and surgeons are in a strategic position as recruitment agents for new nurses.

On June 15, 1943, the U. S. Cadet Nurse Corps was created by unanimous vote in both houses of Congress, to be administered by the U. S. Public Health Service, for the purpose of recruiting 125,000 new student nurses in a two-year period. In its first year the Corps exceeded its goal of 65,000 new nurses by more than 500. This partially relieved the acute nurse shortage. However, 65,000 new nurses will not be enough to combat shortages which still exist in every branch of nursing. In the coming year the additional 60,000 must be recruited if adequate health care is to be given our military and civilian population.

Physicians and surgeons have been seriously concerned with the nurse shortage. Their realization of the need should insure their whole-hearted cooperation with the Cadet Nurse Corps program and their support in recruiting new students. Recognizing the fact that if a recruitment campaign for new student nurses is to be effective it must have the participation of all interested groups, the American College of Surgeons has been enthusiastic in its cooperation. Through this organization, physicians and surgeons have been urged in every possible way to coordinate their efforts with the nursing profession, hospitals and lay organizations in disseminating information about the Corps program.

At present the Corps fulfills three functions. It offers the means of recruiting new student nurses, prepares them more rapidly under the accelerated program, and provides technic for distributing nursing service, a system whereby everyone benefits, including physicians and surgeons, hospitals, the new student nurses and the public.

As a physician or surgeon your contacts are valuable to the program because you have the trust and confidence of your patients and you are often asked for advice by young women interested in nursing. By giving full information about the Corps program and by directing these prospective applicants through the proper channels to the school of their choice, you can help build up the Corps.

Your aid is needed more than ever as the Corps enters its second year. Every physician and surgeon realizes that he is a potential recruitment agent for a program in which his own welfare is vitally concerned.

The Louisiana Nursing Council for War Service earnestly asks your sincere support and effort in securing our state's quota of new student nurses and your words of encouragement for the young women in your hospitals who wear the gray and red of the U. S. Cadet Nurse Corps.

INFECTIOUS DISEASES IN LOUISIANA

The morbidity report of the Louisiana State Department of Health showed that for the first week in January there were reported the following diseases in numbers greater than 10: Pulmonary tuberculosis 46 cases, malaria 23, unclassified pneumonia, influenza, and chickenpox 21 each, with 13 cases of scarlet fever. One case of poliomyelitis was found in Lafayette Parish this week, and eight cases of typhus fever scattered all over the state. For the next week which came to a close on January 13 pulmonary tuberculosis again led reportable diseases with 45 cases, followed by 36 of malaria, 31 of chickenpox, 21 of scarlet fever, 20 of mumps, 18 of measles, and

15 of unclassified pneumonia. Most of the cases of malaria, 34 in number, were reported from military sources. For the week which ended on January 20 again pulmonary tuberculosis led all other reportable diseases with 39 instances, followed by 15 of scarlet fever, 12 of malaria, 11 of measles, and 10 of unclassified pneumonia. This was a remarkably quiet month, evidently logically speaking, as all of the contagious and infectious diseases were remarkably low in their incidence. For the week which came to an end January 27, there were reported the following diseases in numbers greater than 10: Pulmonary tuberculosis 39, scarlet fever and malaria 26 each, unclassified pneumonia 19, chickenpox 17 and influenza 12. The week which finished the month of January showed the inclusion of the accumulated venereal disease reports for the month. This will be first given. There were listed 1,517 cases of gonorrhea, 864 of syphilis, 29 of chancroid, 15 of lymphopathia venereum, and 11 of granuloma inguinale. About half of the cases of gonorrhea came from military sources as did 96 of the cases of syphilis. The other reportable diseases listed this month include 57 cases of chickenpox, 45 of malaria, 39 of pulmonary tuberculosis, 14 each of scarlet fever and unclassified pneumonia, 13 of measles, and 10 of mumps. Forty-five of the cases of malaria were reported from military sources, and in 28 of these reported cases the malaria was contracted outside the Continental United States.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce reported that during the week ending January 13 there occurred in the City of New Orleans 154 deaths, many more than the week previous when only 115 of our citizenry expired. The division between the two races was 102 white, 52 non-white, with 13 of these deaths occurring in children under one year of age. For the succeeding week the number of deaths was 160, of which 106 were white and 54 were colored, with six infant deaths. The week which closed January 27 showed that 162 people died in the City of New Orleans, 100 of whom were white and 62 were colored, and in this total deaths there were nine children under one year of age. This corresponds very closely to a three-year average in the corresponding week in the year. For the week which came to a close on February 3 the 152 deaths in the city were apportioned 104 white, 48 colored, with 12 deaths in children under one year of age.

MONTHLY STATISTICAL REPORT JANUARY, 1945

Estimated Population as of July 1, 1944

White	391,000
Colored	169,000
Total	560,000
Total deaths, all causes	694
White	452
Colored	242
Resident deaths, all causes	363
White	375
Colored	188

DEATH RATES

(Per 1000 per annum for the month)

	All Deaths	Non-residents excluded
White	13.8	11.5
Colored	17.2	13.3
Total	14.8	12.1

Total births recorded	1,460
White	991
Colored	469
Resident births	1,149
White	790
Colored	359

BIRTH RATES

(Per 1000 per annum for the month)

	All Births	Non-residents excluded
White	30.4	24.2
Colored	33.3	23.7
Total	31.3	23.9

DR. WESTON P. MILLER
(1886-1944)

Dr. Wester Peter Miller of Eunice, Louisiana, was born in 1886 and graduated from the School of Medicine, Tulane University, in 1909. Dr. Miller died in Touro Infirmary at New Orleans on October 28, 1944.

DR. CHARLES Y. SEAGLE
(1877-1945)

Dr. Charles Y. Seagle of Bertrandville died at Baptist Hospital, New Orleans on January 29, 1945, at the age of 67. He practiced for many years in Plaquemines and St. Bernard parishes and was the only physician from Arabia to Pointe a la Hache. The difficulties encountered in Dr. Seagle's area were great. He had his own ferry boat to get to the other side of the river. He had to do most of his traveling on horseback and was said to have kept horses scattered around various places so that he could get a fresh one when needed on a long trip. Men such as Dr. Seagle are few and far between. It will be hard to replace him professionally and in the love of his swamp-living clientele.

BOOK REVIEWS

Familial Susceptibility to Tuberculosis: By Ruth Rice Puffer, Dr. P. H. Cambridge, Harvard Univ. Pr., 1944. pp. 106. Price, $2.00.

This small book is essentially a thesis submitted to the Harvard School of Public Health and based upon the studies of the epidemiology of tuberculosis carried on for eleven years in Williamson County by the Tennessee Department of Health. It is a thesis in the original meaning of the word, maintaining the proposition that heredity plays as important a part in the development of tuberculosis as does infection. Dr. Puffer reviews the previous work in this field thoroughly and then gives a statistical analysis of the results of the Williamson County study of twins, siblings, marital consorts and parents. Numerous tables and charts are used to condense these results.

Some very interesting and practical conclusions are drawn. The decline in tuberculosis mortality during the past century is statistically explained by the gradual elimination of susceptible families. Marital tuberculosis is thoroughly discussed and the danger of marriages between individuals with a family history of tuberculosis is pointed out. The importance of a lifelong study of the children of tuberculous parents is clearly demonstrated. Finally, the application of this concept of familial susceptibility to tuberculosis control is outlined to point out the most economical field for case finding in the prevention of this disease. For the statistician there is an appendix with more detailed tables and the statistical methods used. There are a good bibliography and an index. Those interested in the epidemiology of tuberculosis and its control will find this a thought provoking and valuable book.

J. L. WILSON, M. D.

A Bibliography of Aviation Medicine, Supplement; comp. By Phebe Margaret Hoff, Ebbe Curtis Hoff, and John Farquhar Fulton. Washington, D. C., National Research Council, 1944. pp. 109. Price, $2.00.

This supplement continues the splendid work of the authors in bringing together the pertinent references to articles published during the two years since the original Bibliography was published. The authors point out in their preface that the literature of the past two years indicates an emphasis on practical problems. This has necessitated the inclusion of a new subheading on "Survival and Rescue", many new references dealing with the transport of sick and wounded by air. A new subheading on "Rehabilitation" has also been added. Since aviation medicine overlaps so many fields, the list of references includes many which should be of interest and value to the general practitioner as well as to the investigator.

H. S. MAYERSON, Ph. D.

Manual of Clinical Mycology: By Norman F. Conant, Ph. D., Donald S. Martin, M. D., David T. Smith, M. D., Roger D. Baker, M. D., and Jasper L. Callaway, M. D. Philadelphia, W. B. Saunders Co., 1944. pp. 348. Price, $3.50.

This new manual on human diseases due to fungi, prepared by a group of well-known Duke University workers, summarizes admirably and compactly the salient features of modern knowledge concerning both clinical and laboratory aspects of the subject. It is a most valuable addition to the existing literature in the field and should have a deservedly large circulation, particularly among those who are not experts but who require a useful source of reference for aid in the study of suspected mycoses. Military medical officers working in regions where such infections may be expected to occur with any considerable frequency will undoubtedly be grateful for the appearance of this volume. It should also fill the long-felt need for a text which can be recommended to medical students equally when they are beginning to study clinical mycology and later when they are about to enter private practice.

The early chapters deal with the generalized mycoses and those affecting the deeper tissues of the body. The discussion of each type of infection includes a summary of its known geographic distribution; symptomatology; verbal, photographic and x-ray description of the gross lesions; pathology seen at biopsy and at autopsy; photomicrographs of sections of affected tissues; differential diagnosis with the aid of clinical signs, cultural methods or animal inoculations; photographs of distinctive microscopic features and characteristic giant colonies of the etiologic fungus on artificial medium; immunology; prognosis and treatment of the disease.

Subsequent chapters deal with the superficial dermatomycoses, beginning with a general discussion of their symptomatology; prognosis; treatment; immunology and mycology. There follows a more detailed analysis of the important infections in this group, utilizing a method of presentation similar to that for the systemic mycoses. Finally there are chapters on the fundamentals of elementary mycology and methods for the recognition of fungus contaminants, as well as a brief but adequate appendix containing a summary of useful procedures and materials for the study and treatment of the various clinical conditions.

Each of the authors has contributed a discussion of those particular aspects of the various infections in which he is expert and the material has been presented with a uniformity which contributes greatly to the excellence of the book as a whole. The publishers have also done a splendid job in reproducing the abundant photographic material and in setting up the volume so that it can be

handled conveniently. One of the few ways in which this manual might be further improved would be the substitution of colored plates for black and white illustrations in reproducing the features of the lesions and cultural characteristics of the fungi. This however would undoubtedly add considerably to the expense of the book and we can be very grateful for the high quality and reasonable price of the volume as now published.

MORRIS F. SHAFFER, Ph. D.

Etiology, Diagnosis and Treatment of Amebiasis: By Charles Franklin Craig, M. D., M. A. (Hon.), F. A. C. S., F. A. C. P. Baltimore, The Williams & Wilkins Company, 1944. pp. 332. Price, $4.50.

This volume is substantially a revision of the author's work entitled "*Amebiasis, and Amebic Dysentery*", which was published in 1934. The volume consists of twelve chapters under the following headings: Introduction; The Etiology of Amebiasis and Amebic Dysentery; The Epidemiology of Amebiasis; The Pathology of Amebiasis; The Pathology of Amebiasis (Continued); The Symptomatology of Amebiasis; The Complications and Sequelae of Amebiasis; Clinical Diagnosis of Amebiasis: Microscopical and Cultural; Laboratory Diagnosis of Amebiasis: Serological; The Prognosis and Prophylaxis of Amebiasis; The Treatment of Amebiasis.

The author brings to bear on the subject a rich experience and sound judgment that leave no ground for criticism and little for comment. At the end of the volume is a very complete list of references and an author's index. A modest number of well chosen illustrations serve to illustrate special features. Among the many points of special interest to the reviewer is the emphasis laid on the possibility of missing the *Endamoeba histolytica* in a single examination. The author lays special emphasis on the importance of adequately-trained laboratory personnel if the results of the laboratory examinations are to have any value. Col. Craig believes that there is a difference in virulence among the strains of the organism and that those in the tropics are more virulent than are those found in temperate climates. In agreement with the views of most men who have had experience with the amebiasis among military personnel, flies are regarded as important transmitting agents. The author considers that the importance of lower animals as sources of infection may be worth further investigation. He is clearly of the opinion that a tropical climate induces lower resistance of the human carrier. It is gratifying to find stress laid on the importance of considering amebiasis when a diagnosis of appendicitis is being weighed, a feature often overlooked by clinicians. On the important subject of food handlers as possible sources of infection, while the author regards examination and elimination of carriers as important, he considers this can well be carried out only in special groups. He regards "routine examination of all food handlers and their proper treatment as an ideal impossible of attaining and economically most costly."

One is pleased to find so distinguished an authority advising against surgical procedures, appendicostomy and cecostomy as inadvisable. The final paragraph of the book is a plea for better understanding and practice from the point of view of public health, a very fitting conclusion for so important and authoritative a work.

G. W. McCOY, M. D.

PUBLICATIONS RECEIVED

Lea & Febiger, Philadelphia: Internal Medicine, Its Theory and Practice, Edited by John H. Musser, B. S., M. D., F. A. C. P.

D. Appleton-Century Company, New York and London: Approved Laboratory Technic, by John A. Kolmer, M. S., M. D., Dr. P. H., Sc.D., LL.D., L. H. D., F. A. C. P.

The Williams & Wilkins Company, Baltimore: The Abortion Problem, Proceedings of the Conference Held Under the Auspices of the National Committee on Maternal Health, Inc.

Paul B. Hoeber, Inc., New York and London: Anatomy As a Basis for Medical and Dental Practice, by Donald Mainland, M. B., Ch. B., D. Sc., F. R. S. E., F. R. S. C.

The Commonwealth Fund, New York: American Medical Practice in the Perspectives of a Century, by Bernhard J. Stern, Ph. D. Patients Have Families, by Henry B. Richardson, M. D., F. A. C. P.

The Philosophical Library of New York, New York: Modern Methods of Amputation, by Edmundo Vasconcelos, with an Introductory Survey of The Development of Amputation by Major General Norman T. Kirk, M. C.

The Jaques Cattell Press, Lancaster, Pa.: The Marihuana Problem in the City of New York, by the Mayor's Committee on Marihuana.

Chemical Publishing Company, Inc., Brooklyn, N. Y.: The Chemistry and Pharmacy of Vegetable Drugs, by Noel L. Allport, F. I. C.

Charles C. Thomas, Springfield, Illinois: Poet Physicians, An Anthology of Medical Poetry Written by Physicians, compiled by Mary Lou McDonough.

University of Michigan, Ann Arbor, Michigan: Technique of the Standard Kahn Test and of Special Kahn Procedures, by Reuben L. Kahn.

College Book Company, Columbus, Ohio: A Test for Color Blindness, by P. B. Wiltberger, M. D.